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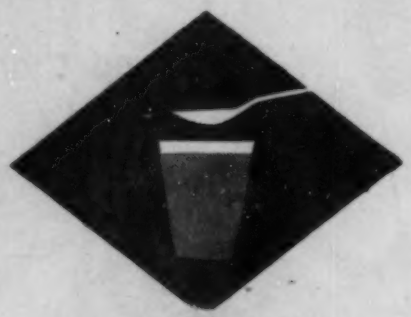

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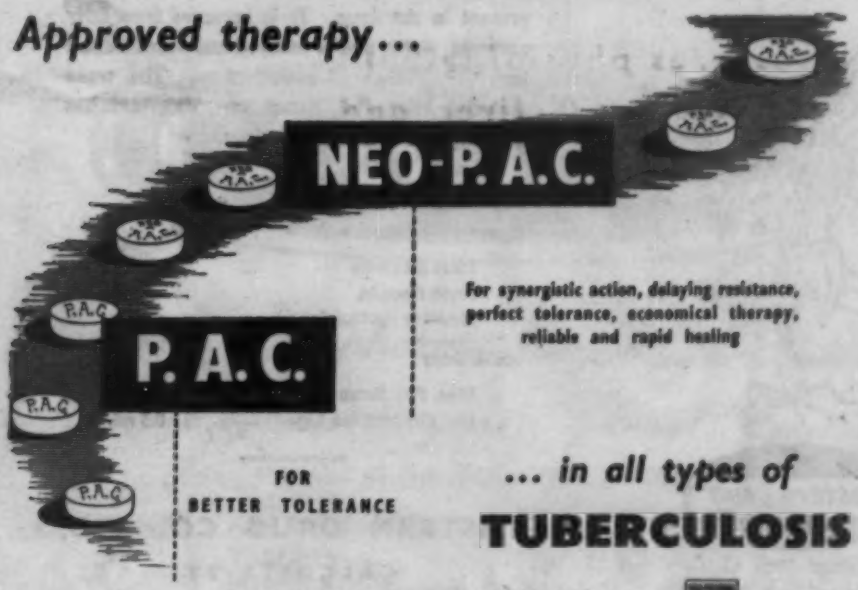

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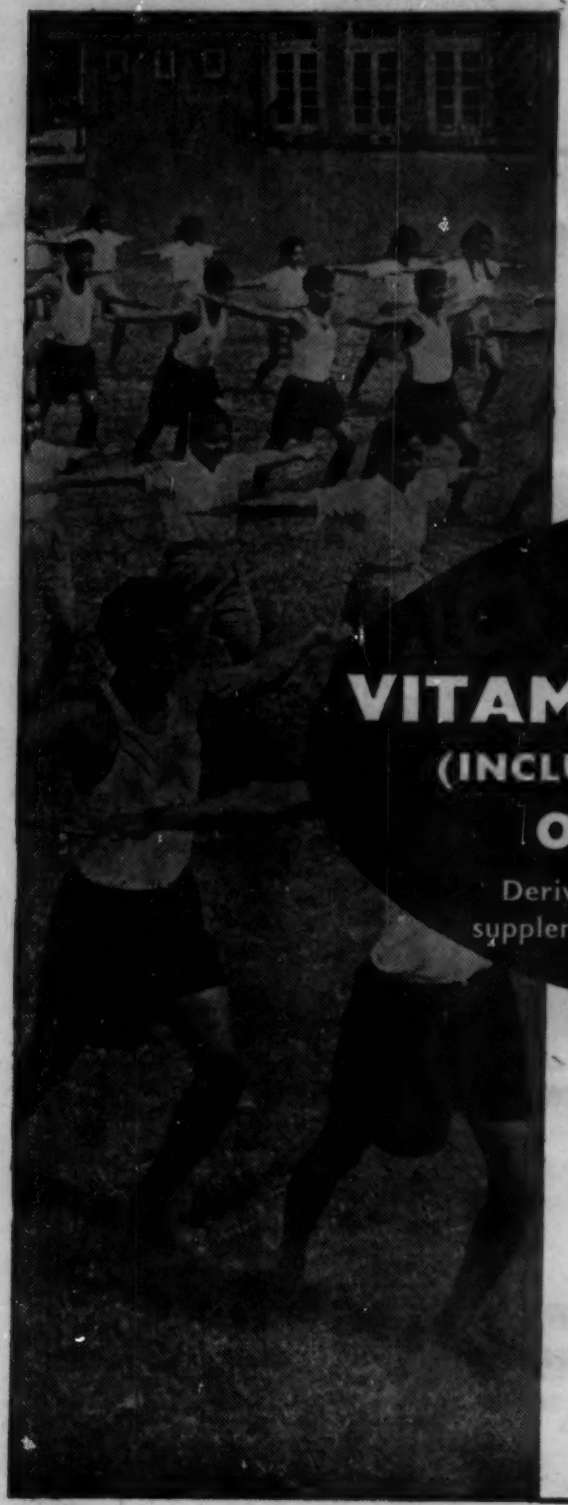

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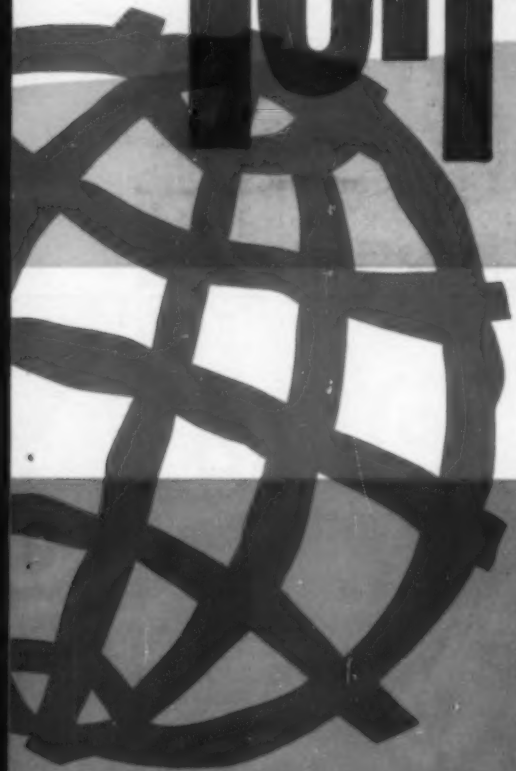

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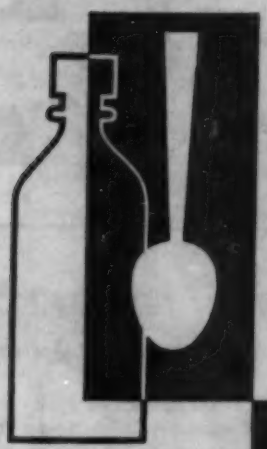

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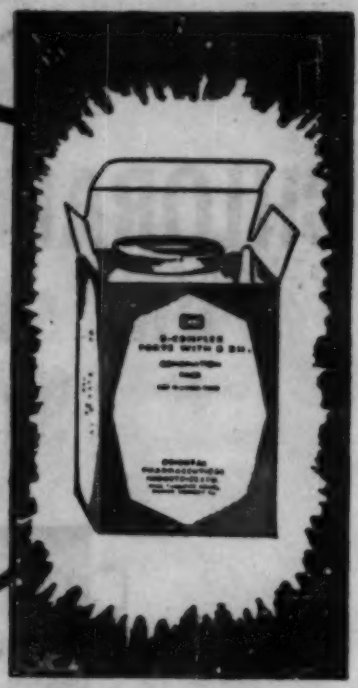

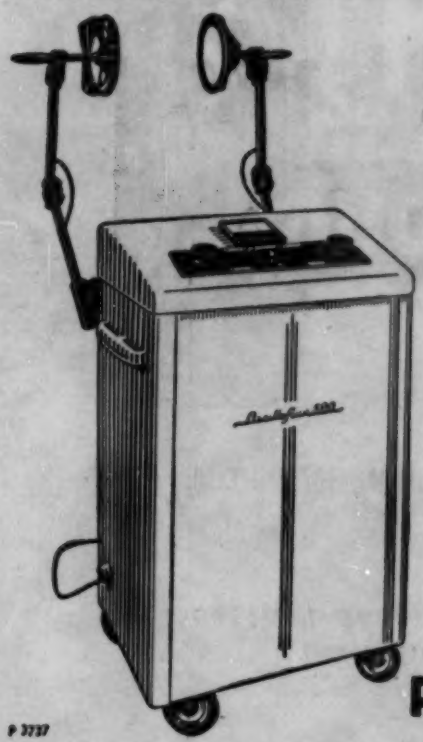

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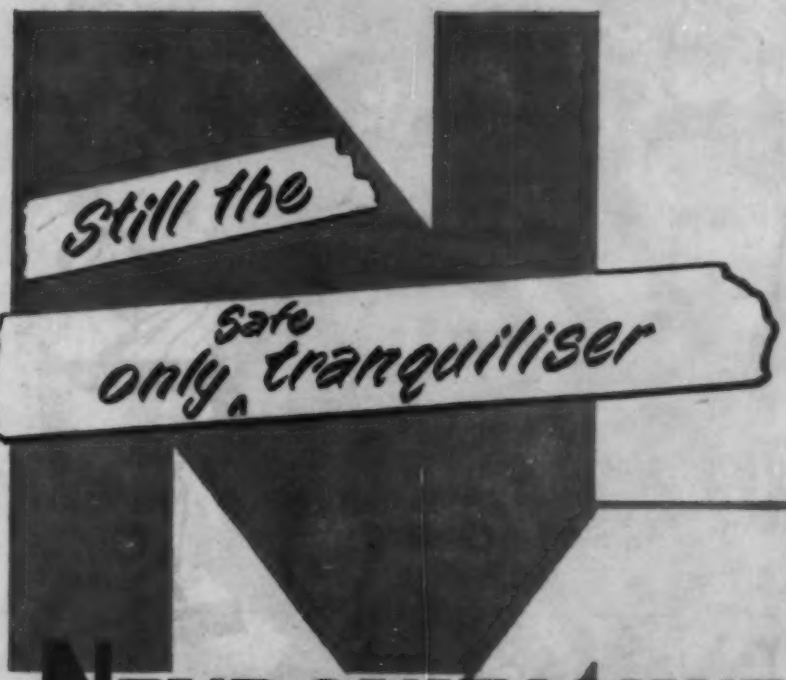

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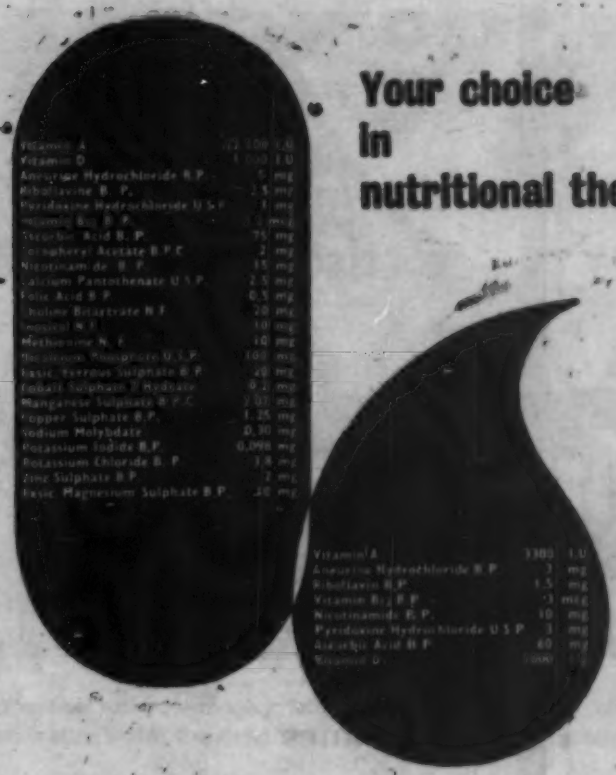

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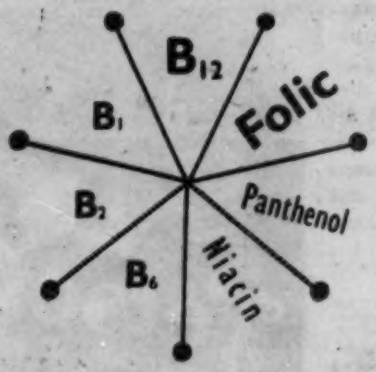

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CALCUTTA
FEBRUARY 16, 1960

ORIGINAL ARTICLES

INFANTILE CIRRHOSIS

A Clinicopathological Study of Forty-one Cases

J. N. BERRY, M.D., M.R.C.P., D.C.H. (LOND.) AND G. S. SAINANI, M.D. (NAGPUR)

Department of Medicine, Medical College, Nagpur

INTRODUCTION

The pathogenesis of infantile cirrhosis is still a mystery. Much work has been done recently but still the goal seems to be hardly within sight. There is no known set of dietary conditions which produce in the experimental animals the sequences seen in man. Several aetiological features have been suggested by different workers and the subject has been exhaustively reviewed by the Liver Diseases Sub-committee, Indian Council of Medical Research (1955). This study was done with a view to find out the probable aetiological factors working in our cases and also to correlate the clinical features with functional and histological findings.

METHODS AND MATERIAL

A detailed clinical, laboratory and histological study was undertaken in 41 cases of infantile cirrhosis admitted in the Medical College Hospital, Nagpur during the period of 2 years from 1st July 1955 to 30th June 1957.

The cases were studied according to the pro forma based on 'clinical case sheet for investigation on cirrhosis liver' recommended by Indian Council of Medical Research with a few additions and modifications.

Needle biopsy of liver was performed by Vim Silverman's needle and the procedure followed was that described by Wahi (1946). The specimens of liver obtained were studied for the degree of liver cell damage and were categorised into "early stage," "well established stage," "very advanced and late stage," as adopted by Shrivastava and Aikat (1954).

OBSERVATIONS

I. CLINICAL FEATURES :

(i) *Age and sex*—The age and sex distribution of 41 cases is given in Table 1.

TABLE 1—SHOWING AGE AND SEX INCIDENCE

Age	Male	Female	Total
Under 1 year ...	0	0	0
1—2 years ...	16	6	22
2—3 years ...	8	1	9
3 years and above ...	7	3	10
Total ...	31	10	41

(ii) *Religion and caste*—All the cases belonged to Hindu religion ; out of 41 cases, 32 were from orthodox vegetarian families and the rest (9) were from backward and scheduled castes. The economic status of only the later 9 cases was poor.

(iii) *Onset of disease*—It was insidious in all cases and in the majority of them it started with anorexia, gastrointestinal disorders, irregular fever, irritability and prominence of abdomen. Jaundice, swelling of the feet and marked disten-

sion of the abdomen were late features. In no case was there any history of fever with jaundice preceding the present complaints.

(iv) *Details of mother's history*—Mother's health was good in 12 cases, average in 20 cases and poor in 7 cases while in 2 cases the mother was not alive.

The mothers' health during pregnancy was fairly satisfactory in all and in no case was there any history of jaundice during pregnancy.

Out of 41 cases, 35 were strict vegetarians, the staple diet of the mother being wheat, rice, pulses, vegetables and sometimes fruits and milk. The remaining 6 cases gave a history of taking meat once a week or so.

The analysis of the incidence of the disease in different birth orders is given in Table 2.

TABLE 2—SHOWING RELATIONSHIP BETWEEN THE INCIDENCE OF THE DISEASE AND BIRTH ORDERS OF THE PATIENTS

Birth order of patients	No. of cases	
	Observed	Expected
1st	15	8·7
2nd	9	8·1
3rd	4	7·0
4th	5	5·7
5th	4	4·0
6th	3	2·6
7th	1	1·9
8th	1	1·2
9th and above	0	1·8
Total ...	41	41·0

In this series, the firstborns predominate. But in the general population also the number of first-born children are more numerous than the children of any other birth order. Therefore, from the figures of all live births in Nagpur in 1955, the expected number of children in different birth orders was calculated. Even in the expected number the maximum frequency is in the 1st birth order but much less than the observed figure which clearly shows that the incidence of the disease is more common in the first sibling.

History of liver disease in previous siblings was present in 4, all of whom had died of it. One case was interesting in this series in that the person had two wives—all the five issues by the first wife were healthy while 2 issues by the second wife suffered from infantile cirrhosis. This may be a coincidence but any multiplication of such examples shall favour the assumption of a noxious agent, possibly virus, spreading from the mother to the infant, prenatal or postnatal.

(v) *Infant's feeding and dietetic history*—4 cases had cow's milk alone (breast feeding being insufficient or contraindicated). 13 cases had breast feeding and cow's milk to supplement the breast feeding, started a few months after birth and 24 cases had breast feeding till weaning and cow's milk with usual vegetarian diet (rice and dal) after weaning.

(vi) *Symptoms at onset*—Symptoms with which the patients came when admitted are arranged according to the frequency:

1.	Swelling of abdomen	32 cases
2.	Low grade fever	27 „
3.	Jaundice	26 „
4.	Oedema feet	18 „
5.	Irritability	14 „
6.	Epistaxis	10 „
7.	Loss of appetite	8 „
8.	Pruritus	3 „
9.	Bleeding per rectum	2 „
10.	Loss of weight	7 „
11.	Unconsciousness	1 case

(vii) *Physical signs*—Liver. Except in one case was palpable in all. Enlargement varied from 1/4″ to 3″ below the right costal margin in midclavicular plane. The consistency was firm and surface smooth in practically all while the border was sharp in the majority of cases.

Spleen. A firm spleen was palpable from 1/4″ to 4″ below the left costal margin in 31 out of 41 cases (75·6 per cent).

Ascites and abdominal veins. Ascites was present in 27 out of 41 cases (65·8 per cent). Abdominal veins were prominent in 23 cases and the direction of flow of blood was from below upwards in 19 cases (possibly due to ascites causing pressure on inferior vena cava) and away from umbilicus in 4 cases.

Other features. Palmar erythema was present in 7 out of 41 cases (17 per cent). Marked irritability was observed in 19 cases (46·3 per cent) and signs of incipient hepatic coma in 5 cases.

II. LABORATORY INVESTIGATIONS:

All the cases showed some degree of anaemia which was dimorphic or microcytic. In only 2 cases it was macrocytic. E.S.R. was raised in 76 per cent of cases. Blood for Kahn's test was examined in 23 cases in both mother and infant. In no case was it positive in child and mother at the same time. Separately it was positive in 2 infants and 8 mothers; the remaining were negative. In the Kahn test positive infants, the histological features of the liver were not those of syphilitic cirrhosis.

On urine examination bile pigments were present in 26 cases, bile salts in 10 cases and excessive urobilinogen in 3 cases.

Typical clay-coloured stools were seen in 8 cases. No ova or cysts were found in stools.

Ascitic fluid was a transudate in all cases with ascites.

Liver function tests—These were done in all the 41 cases and results of these were classified according to the parenchymal damage as shown by flocculation tests and albumin/globulin ratio estimation:

(1) Marked parenchymal dysfunction when thymol turbidity 11 units or above or thymol flocculation +|+ + + and/or A/G (albumin/globulin) ratio less than 1.

(2) Moderate parenchymal dysfunction when thymol turbidity between 5 and 10 units or thymol flocculation + + or +|+|+ and/or A/G ratio between 1 and 1·5.

(3) No parenchymal dysfunction when thymol turbidity between 1 and 4 units and flocculation 0 or + and A/G ratio more than 1·5.

According to this classification, the degree of liver parenchymal damage as judged functionally was as follows:

Degree of parenchymal dysfunction	No. of cases	Per cent
1. Marked	8	19·5
2. Moderate	23	56·1
3. Nil	10	24·4
Total ...	41	

III. HISTOLOGICAL EXAMINATION:

Needle biopsy of the liver was successfully performed in 25 out of 41 cases. Majority of the cases on whom the needle biopsy was done were clinically well-established cases of cirrhosis liver. The sections were graded as follows: Early stage (Grade I)—4 cases; well-established stage (Grade II)—15 cases; very advanced and late stage (Grade III)—5 cases; normal liver appearance—1 case only (Figs. 1, 2, 3 and 4, *vide* Plate).

Correlating the presenting clinical features with different degrees of liver cell damage, some interesting clinical inferences were drawn:

1. All the 4 early stage cases, as reported by the pathologist, were labelled clinically also as early cirrhosis. The constant finding in this group was a firm palpable liver with sharp lower border but only one case had mild ascites and jaundice.

2. Out of the 15 Grade II cases, 5 cases were diagnosed clinically as early cirrhosis as there was no ascites and out of the 5 late stage cases, one case was clinically in early stage as there was mild ascites and no jaundice.

3. Liver was palpable in all the stages of liver cell damage and there was not much difference in the size of the liver enlargement in different stages.

4. The frequency of fever, jaundice, oedema of the feet, ascites, splenic enlargement, prominent abdominal veins, palmar erythema and irritability increased with the degree of liver cell damage.

The different grades of liver cell damage as shown by the needle biopsy have been correlated with the results of liver function tests in Table 3 and Fig. 5.

TABLE 3—SHOWING COMPARISON OF LIVER FUNCTION TESTS IN DIFFERENT GRADES OF LIVER CELL DAMAGE IN THE CASES OF CIRRHOSIS IN INFANTS

Tests	Grade 0 (1 case)	Grade I (4 cases)		Grade II (15 cases)		Grade III (5 cases)	
		Mean	Range	Mean	Range	Mean	Range
Serum protein (g. %)	6·5	6·2	6·0-6·6	5·1	3·0-6·8	4·7	3·6- 5·6
Serum albumin (g. %) ...	4·0	4·4	4·1-4·8	2·9	1·6-4·4	2·7	2·1- 3·1
Serum globulin (g. %) ...	2·5	1·9	1·5-2·5	2·3	1·0-3·7	1·9	0·6- 2·6
A/G ratio	1·6	2·3	1·9-2·7	1·3	1·0-1·6	0·9	0·8- 1·3
Thymol turbidity (units) ...	6·0	5·6	4·0-6·0	6·8	4·0-8·0	6·4	4·0- 8·0
Thymol flocculation	2·0	1·2	0·0-2·0	2·2	0·0-3·0	2·0	0·0- 3·0
Zinc turbidity (units)	16·0	12·00	8·0-14	14·6	10·0-18	18·0	16·0-20·0
Serum bilirubin (mg. %) ...	1·25	0·6	0·4-0·8	7·6	0·4-26	9·0	0·6-12·0
Serum alkaline phosphatase (units B)	8·6	4·8	2·0-5·0	5·3	3·2-9·6	7·5	4·2-11·5
Prothrombin index (%) ...	—	95·3	74-100	57·6	43·5-83·7	48·5	43·7-91·6
E.S.R. (mm., Wintrobe) ...	28	20·3	15-28	36·9	30-62	20·4	20 -62

FIG. 5—SHOWING COMPARISON OF LIVER FUNCTION TESTS IN DIFFERENT GRADES OF LIVER CELL DAMAGE IN INFANTS

DISCUSSION

Aetiological features—Infantile cirrhosis is fairly common in this part of the country. Out of 41 cases, 31 (75·6 per cent) were in the age group of 1 to 3 years. This finding is quite consistent with the reports of other workers from India (Achar, 1955 ; Shrivastava and Aikat, 1954). All workers, except Achar and Raju (1951), report a higher incidence of infantile cirrhosis amongst male children and our observations also reveal that out of 41 cases, 31 were males.

From whatever reports that are available in India, it is evident that the disease is restricted to Hindus. Amongst Hindus again the children of particular classes of people, like Brahmins, are the maximum sufferers. All our cases belonged to Hindu religion and out of 41 cases, 32 were drawn from orthodox vegetarian families, mainly Brahmins.

On analysing the economic status of the parents of these cases, it was found that 79 per-cent cases belonged to middle and rich classes. Other workers (Achar and Raju, 1951 ; Mukherjee, 1952 ; Shrivastava and Aikat, 1954) also report a higher incidence in rich and middle class people but Vyas (1955), working in Saurastra, observed that the majority of his cases belonged to the poor class people.

It is unanimously agreed that the disease has a tendency to run in successive children in a family, although the percentage of such occurrence varies. Achar (1955) obtained such a history among siblings in 12 per cent of his cases in Madras, while Mukherjee (1952) from Indore found such incidence in 26 per cent of his cases, and Shrivastava and Aikat (1954) from Gwalior encountered it in 20 per cent of their cases. We in our series of 41 cases got such history in 4 cases (9·8 per cent) only. No good reason is given so far to explain the occurrence in successive children. Transfer of toxin and/or organisms from the mother to the foetus across the placenta has been postulated (Kini, 1940). In such familial instances, Wahi (1949) suggested that the cause might be a virus infection which the infant had contracted from the mother's infected genital tract during birth. In our cases there was no history of jaundice during pregnancy in mothers of these children but this does not rule out the possibility of subclinical infection in the mother. Recently it is claimed by some workers (Mukherjee, 1952 ; Srivastava and Aikat, 1954) that there is a higher incidence of the disease in the first child. Our observations also point to the same fact because out of 41 cases, 15 cases belonged to the first birth order. The frequency of the disease decreased as the birth order increased.

Shrivastava and Aikat (1954) and Vyas (1955) found that all their cases belonged to vegetarian families but in our series there were 6 cases from families who were meat eaters. Much has been written about the prevalence of the disease among children belonging to vegetarian families and this has been thought to be in favour of nutritional

actiology. Vegetarianism or non-vegetarianism carries no meaning about the adequacy of the diet unless it is accompanied by an estimate of the quantity and quality of the diet to rule out malnutrition in the form of an unbalanced diet in infantile cirrhosis. From whatever reports are available so far, except that of Vyas (1955), it is clear that the disease is not associated with extreme poverty.

There is nothing much to comment on the infant's feeding and dietetic history of our cases as the majority of them (24 cases) conformed to the usual practice of giving breast feeding till weaning and cow's milk with vegetarian diet after weaning.

Role of infection in causation of infantile cirrhosis was stressed by Harrel and McBryde (1940). In recent studies of infantile cirrhosis (Achar and Chacko, 1954), a history of transient jaundice could be elicited in a fairly large proportion of cases. In considering the problem of hepatitis, a World Health Organisation Committee recommended that the relation of cirrhosis in children to virus hepatitis requires further study. Congenital syphilis was blamed at one time as the causative factor of cirrhosis in children. Considering the above factors in our cases, we find that there was no evidence of any infection which could have played a causative role in our series.

Clinical features—Onset of the disease was insidious in all cases. Swelling of the abdomen was the presenting feature and was manifest in about 80 per cent of cases. Low grade fever (65·8 per cent) and jaundice (63·4 per cent) were also commonly encountered as the majority of our cases were in a well-established stage.

Amongst the physical findings, palpable liver was a constant feature—it was palpable in all except one. Ascites was present in 27 out of 41 cases (65·8 per cent); amongst these 27 cases of ascites, splenic enlargement was observed in 24 cases (88·8 per cent), prominent abdominal veins in 26 cases (77·0 per cent). In contrast in the 14 cases without ascites, spleen was palpable in 7 cases (50 per cent) and abdominal veins were prominent in 3 cases (21·4 per cent). It seems from this clinical correlation that portal hypertension has some contributory role in the causation of ascites as clinical features like splenomegaly and prominent abdominal veins occurred with greater frequency in cases with ascites than without ascites.

On the basis of thymol turbidity, thymol flocculation and albumin/globulin ratio estimation, the degree of parenchymal dysfunction was estimated in each case. Summarising all these, we find that 75·69 per cent cases show evidence of parenchymal dysfunction and 24·4 per cent do not reveal such evidence, though clinically they have features of cirrhosis liver.

Comparing the functional with the structural disturbances as represented in Table 3 and Fig. 5, it is evident that serum albumin, serum globulin, A/G ratio, thymol turbidity and flocculation, serum bilirubin and serum alkaline phosphatase show increasing abnormality as the degree of liver cell damage increases. Total protein, zinc turbidity and E.S.R. do not show proportionate abnormality as in the above tests. Further, it is clear that as the degree of liver damage increases, the number of liver function tests showing abnormality increases.

Correlating the clinical features with different grades of liver cell damage in infantile cirrhosis, we found that there is no absolute correlation between the two. We find that four Grade I cases were diagnosed clinically also as early cirrhosis but of 15 Grade II cases, 5 were diagnosed clinically as early cirrhosis and out of 5 Grade III cases, one was more towards the early stage clinically. This shows that though clinically some cases were in an early stage, histologically they were far advanced. There was no difference in the size of the liver in these 3 grades of infantile cirrhosis. Liver has been reported to diminish in size as the disease advances but such was not seen in our cases. The frequency of clinical features like fever, jaundice, ascites, splenic enlargement, prominent abdominal veins, oedema of the feet palmar erythema and irritability increased with the degree of liver cell damage.

Summary and Conclusions

Forty-one consecutive cases of infantile cirrhosis, admitted from 1st July 1955 to 30th June 1957, have been studied clinically, functionally and histologically. Aetiological features have been discussed. The relative frequency of important symptoms and signs like ascites, jaundice, splenomegaly and prominent abdominal veins have been assessed and their interrelationship discussed. Correlations between clinical features with structural changes and functional alterations with structural changes have been studied.

The conclusions drawn from this study are as follows:

The incidence of infantile cirrhosis of the liver is highest in the age group 1—3 years and the disease is more common in male children. In this series of 41 cases, the disease was restricted to Hindus and was more in the orthodox class of people, like Brahmins, and in children belonging

to the rich and middle class families. The disease has a tendency to run in successive children in a family and its incidence is highest in the first child as seen from comparison of the observed and the expected figures in our series. No definite aetiological factor was found in our cases. Our impression is that congenital syphilis, worm infestations etc., do not play any causative role and the cause may be malnutrition in the form of an unbalanced diet or virus infection or both.

Palpable firm liver with sharp lower border in a child of the age group 1-5 years should be taken as the diagnostic lead for infantile cirrhosis.

The commonest type of anaemia as seen in our cases was the dimorphic type. On the whole the anaemia in these cases was not of severe degree.

About 75·6 per cent of cases showed evidence of parenchymal dysfunction and the remaining 24·4 per cent did not reveal such evidence though clinically they had features of cirrhosis liver.

Serum albumin, serum globulin, albumin/globulin ratio, thymol turbidity and flocculation tests, serum bilirubin and serum alkaline phosphatase show increase in abnormality proportionate to the liver cell damage, while total proteins, zinc turbidity and erythrocyte sedimentation rate have no significant correlation with advancing liver damage.

With the increasing degree of liver cell damage, increasing number of liver function tests tend to be abnormal, thus emphasising the advantage of a battery of liver function tests to judge the degree of liver cell damage.

There is no close parallelism between the clinical features and the degree of liver cell damage as seen by the needle biopsy of liver. A clinically early case may show liver cell damage and *vice versa*.

The size of liver enlargement was practically the same in all stages of the disease. The frequency of fever, jaundice, ascites, splenic enlargement, prominent abdominal veins, oedema feet, palmar erythema and irritability increased with the degree of parenchymal damage.

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CORTICOSTEROIDS IN PYOGENIC MENINGITIS

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Pyogenic meningitis is still a serious disease and carries an over-all mortality of 20-35 per cent and a sizable morbidity rate inspite of chemotherapy and antibiotics. Feibush *et al* (1952) quote 70 per cent mortality in their cases. Banks (1954) gives a mortality rate of 22 per cent in cases of meningococcal meningitis treated with sulphonamides and penicillin. Other figures are between 30 and 50 per cent (Bunn and Peabody, 1952; Pengelly, 1955). When corticosteroids became freely available, they were used in pyogenic meningitis just as in various other acute infections. The drug is supposed to diminish the pressure on the brain by reducing the inflammatory oedema. Hypersensitivity to the invading organism is also cut down (Ribble and Abraham, 1958). The results are not very clear, though there are reports on the usefulness of the drug in such cases (Cassidy, 1947; Bernheim *et al*, 1958; Ribble and Abraham, 1958).

This paper deals with a brief review of 50 cases of pyogenic meningitis with special reference to the value of corticosteroids, as per hospital records during the last 3 years.

The cases analysed were between the ages of 13 and 80 years. The patients from the Children Ward were not included. The male to female

ratio was 5:1. They had fever, headache, vomiting, neck rigidity and a positive Kernig's sign. A few had convulsions or hemiplegia and/or cranial nerve palsies. 14 were admitted in coma, 26 were semi-conscious or delirious and 10 were fully conscious. None of them had any skin rash. In a few, ear or skin infection was discovered. One case had associated lobar pneumonia.

In all cases the diagnosis was confirmed by withdrawal of frankly purulent or turbid cerebrospinal fluid under tension. The C.S.F. examination revealed large numbers of pus cells, increased proteins, diminished or absent sugar; organisms were sought for both by smear and culture method. Unfortunately, they were detected only in seven cases, in 4 they were meningococci, in 2 pneumococci and in 1 staphylococci. Most of the patients were admitted in the hospital after 2-3 days of the onset of symptoms and had received inadequate treatment with some antibacterial agent. This could be the cause of such a low incidence of positive cultures in our cases. White cell count was high in all the cases, except in 2 where it could be counted as within normal limits.

Treatment

Immediately on the withdrawal of purulent or turbid C.S.F., the patients were put on the following lines of treatment:

1. Crystalline penicillin i.m. 10 lacs stat, and 5 lacs 6-hourly till the temperature was normal for 3 days and C.S.F. was clearing up, and then tapered off.

2. Sulphadiazine i.v. 2 g. stat. and 1-2 g. i.v. 6-hourly, dose being 5-8 g. a day, later 1-2 g. orally 4-hourly. The daily dose of sulphadiazine was 8-12 g. till the temperature settled and then tapered off in 7-10 days. While the patients were on this treatment, the urine output was carefully watched and urine tested for sulpha crystals. Two cases went into near anuria but both recovered completely.

3. 90 per cent of the patients required i.v. fluids and every patient was given alkalies. Adequate nourishment was provided through Ryle's tube, if needed. In the event of circulatory failure, noradrenaline was added to the i.v. drip.

4. Sedatives, oxygen and other supportive measures were adopted as and when required.

5. Only in 7 cases, intrathecal penicillin was used in dosage of 5,000-20,000 units daily for 2-4 days.

6. Corticosteroids were used in 27 cases out of 50. Preference was given to severe and highly toxic cases. Prednisolone 5-10 mg. 4-or 6-hourly was given. In a few cases i.v. hydrocortisone 100 mg. 6-hourly was used.

7. Broad-spectrum antibiotics were used in 13 cases in the following circumstances:

(a) when a patient on sulphonamides showed renal complications;

(b) when response to penicillin and sulphonamides was not evident.

Results

The cases have been divided into three groups:

(i) Comatose;

(ii) Semi-conscious or delirious; and

(iii) Fully conscious patients.

In each group, comparison has been made between those who had corticosteroids and those who had not, while the basic treatment remained the same in all the cases (Table 1).

Table 1—Showing the Response to Therapy in 3 Groups

State of consciousness	Cases with corticosteroids			Cases without corticosteroids		
	No. of cases	Recovery	Mortality	No. of cases	Recovery	Mortality
Comatose ...	6	2	4	5	3	2
Semi-conscious or delirious ...	10	6	4	15	12	3
Fully conscious ...	7	7	nil	2	2	nil
Total ...	23	15	8	22	17	5

In group 1, 14 cases were comatose, out of which 3 died within 24 hours. They have been excluded on the grounds that enough time was not available for the therapy to be effective. Thus, out of 11 cases, 6 were put on corticosteroids and 4 died. Out of the 5 cases who were treated without corticosteroids, 2 died.

In group 2, of semi-conscious or delirious patients, one case died within 24 hours of admission. 25 cases remained for analysis. Ten cases were put on corticosteroids and 4 died. 15 cases were treated without corticosteroids and only 3 died.

In group 3 of fully conscious patients, there were 10 cases. One left against medical advice. Seven cases had corticosteroids and all recovered. Two cases who did not receive corticosteroids also recovered. Hence in the group of conscious patients, all rececovered whether they received corticosteroids or not.

Out of the 12 cases, where broad-spectrum antibiotics were used, 6 died. Mortality in cases who were given intrathecal penicillin was slightly higher than in the rest.

Discussion

Management of acute pyogenic meningitis has been greatly modified during the past 15 years. Recently corticosteroids have been tried in pyogenic meningitis by various workers (Cassidy, 1947 ; Bernheim *et al*, 1958, Ribble and Abraham, 1958). Ribble and Abraham (*loc. cit.*) used ACTH and corticosteroids in pneumococcal meningitis with favourable results. Bernheim *et al* (1958) used i.v. hydrocortisones in 10 cases of severe meningococcal meningitis. The results of this treatment were considerably better. Menichini (1958) used these hormones in pyogenic meningitis and showed good effect on the initial toxic state, on the collapse, on the convulsions and haemorrhagic manifestations. However, in our cases, corticosteroids, given in addition to penicillin and sulphonamides, did not increase the recovery rate. On the contrary, the mortality rate is higher in the group treated with corticosteroids. No final conclusion can be drawn but the facts are revealing. Singh and Tandon (1959) report on the use of corticosteroids in diphtheria. They conclude that the mortality in the corticosteroids-treated cases was higher as compared to the control cases, the basic treatment of diphtheria being the same in all the cases. Intrathecal penicillin and broad-spectrum antibiotics also did not make any difference in the recovery rate. Intrathecal penicillin has been reported to be harmful by some workers and of no advantage by others, except perhaps in pneumococcal meningitis. It might be mentioned that more than 20,000 units intrathecal penicillin at a time is dangerous ; 5 lacs is reported to be a lethal dose by one British worker (Pengelly, 1955) in whose case it was used through an error.

The over-all mortality in our cases has been 33 per cent. In three corticosteroid-treated group it was 37·5 per cent and without corticosteroids it was 22·5 per cent. Thus, results in our cases have not been encouraging. The patients in the series of Cassidy (1947) and of Menichini (1958) were not comatose ; in our ;series the mortality was higher in the comatose patients and lowest in the conscious patients. Whether intrathecal corticosteroids would be of more value in pyogenic meningitis is still to be assessed though some authors (Didier, 1958, Wisemann and Levy, 1958) report on its beneficial effect.

Summary

Fifty patients between 13 and 80 years of age suffering from pyogenic meningitis are reviewed with a special reference to corticosteroid therapy. The number of unconscious and semi-conscious patients was much higher than those of the fully conscious patients. The mortality rate in the corticosteroid-treated group was higher than that reported by various workers.

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SERUM GLUTAMIC OXALACETIC TRANSAMINASE LEVELS IN CORONARY HEART DISEASE

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In the present article an estimation of serum glutamic oxalacetic transaminase (S.G.O.T.) levels has been done in a series of healthy adults, in some cases with liver disease or dysfunction

and in selected cases of coronary heart disease. Some conclusions regarding the value of S.G.O.T. have been drawn from this study.

The cases of coronary heart disease have been selected on clinical and/or electrocardiographic basis. All these cases had their first clinical attack and the S.G.O.T. level was estimated within first 36 hours. This has been done to get an uncomplicated picture for comparative study.

The coronary disease cases have been classified under the following groups:

(1) Cases in which the E.C.G. showed frank evidence of myocardial infarction. (2). Cases where the clinical picture was suggestive but the E.C.G. showed only equivocal changes, e.g., flat or inverted T-waves in some of the leads. (3). Cases where the clinical picture was fairly characteristic yet the E.C.G. recorded no change, even when repeated twice within 3-4 days' time.

Results

The results are shown in Tables 1, 2, 3, 4 and 5.

Table 1—Showing S.G.O.T. Levels in Healthy Adults (Controls)

No.	Name	Age (in years)	Sex	S.G.O.T. level (in units)
1	J.C.J.	24	M	35
2	I.M.	25	M	38
3	R.C.	27	M	41
4	O.P.	18	M	80
5	M.J.	30	F	23
6	G.	32	M	48
7	R.K.S.	26	M	50
8	J.P.	48	M	52
9	L.W.	40	M	41
10	H.D.	39	F	36

Range of S.G.O.T. levels—23-52 units. Average—41·4 units.

Table 2—Showing S.G.O.T. Levels in Cases with Manifest Liver Disease or Dysfunction

Name	Age (in years)	Sex	Disease	S.G.O.T. level (in units)
C.R. ...	60	M	Cancer liver	67
J.M. ...	14	M	Infective hepatitis	59
S.V. ...	42	M	do.	66
S.L.S. ...	32	M	Cirrhosis liver	62
S.S. ...	30	M	Enlarged liver of congestive heart failure	48

Range of S.G.O.T. levels—48-67 units. Average 61 units.

The case with long standing congestive heart failure due to mitral valvular disease also had an elevated S.G.O.T. This fact may be borne in mind while evaluating the S.G.O.T. level in cases of coronary heart disease with right-sided congestive failure.

The S.G.O.T. level was above 65 units in all the 40 cases. 12 cases (28·8 per cent) had S.G.O.T. level between 65-100 units and 12 cases (28·8 per cent) between 101-150 units. Levels above 150 units were noted in 16 cases out of which 3 had a level above 300 units and 3 above 200 units.

Table 3—Showing S.G.O.T. Levels in 17 Cases where E.C.G. Findings were Typical of Recent Myocardial Infarction

	Range of S.G.O.T. levels (in units)					
	65-100	101-150	151-200	201-250	251-300	301-330
No. of cases	2	5	6	1	1	2
Percentage	11·6	29·0	34·8	5·8	5·8	11·6

Table 4—Showing S.G.O.T. Levels in 15 Cases which Showed Only Equivocal Changes in E.C.G.

	Range of S.G.O.T. levels (in units)					
	65-100	101-150	151-200	201-250	251-300	301-316
No. of cases	10	3	0	1	0	1
Percentage	66	19·8	0	6·6	0	6·6

Most of these cases had S.G.O.T. levels below 150 units. The levels in 66 per cent were between 65-100 units. One case had 218 units and another 316 units. These were presumably cases with gross degree of infarction which the E.C.G. failed to show up in the first 36 hours. Even among the first group with S.G.O.T. levels ranging between 65-100 units it can be said that many of these had myocardial damage which would otherwise have just passed off as ischaemia on the basis of electrocardiogram alone. These cases had no evidence of any other disease of an organ which could explain the raised S.G.O.T. levels.

There was one case in which the S.G.O.T. level was 24 units only and remained at nearly that level on two subsequent estimations.

There was one more case who came in severe shock and died soon after admission before an E.C.G. could be taken. His S.G.O.T. was 110

units. From Table 5 it is obvious that the S.G.O.T. estimation is of definite value in diagnosis even in cases where the E.C.G. is negative.

TABLE 5—SHOWING S.G.O.T. LEVEL IN 8 CASES WHERE THE E.C.G. WAS COMPLETELY NEGATIVE ON TWO SUCCESSIVE OCCASIONS BUT THE CLINICAL PICTURE WAS FAIRLY SUGGESTIVE OF MYOCARDIAL INFARCTIONS

	Range of S.G.O.T. levels (in units)						
	Below 40	65-100	101-150	151-200	201-250	251-300	301-330
No. of cases ...	1	3	2	0	1	1	0
Percentage ...	25	75	50	0	25	25	0

DISCUSSION

It is found that in many cases where either the E.C.G. was normal or showed only ischaemia pattern, S.G.O.T. was helpful in the diagnosis of myocardial infarction. In cases with frank electrocardiographic evidence of myocardial infarction, the S.G.O.T. level was higher than in others. The range of S.G.O.T. level in our series was 65-322 units. In the liver disease cases the range of S.G.O.T. level was 52-67 units with an average of 61 units. The normal controls had an average level of 41·4 units of S.G.O.T.

S.G.O.T. estimation is assuming a definite role in the study of coronary artery disease especially in diagnosing some cases where E.C.G. is normal during the first two days of an attack and in the diagnosis of any extension of the thrombus in the coronary artery when the patient is already under treatment and thus helping to judge the prognosis of a case.

In the end however, it must be added that the test is no answer to clinical diagnosis of the disease. It only helps in certain circumstances.

SUMMARY

A study of the S.G.O.T. level has been made in controls, in a few cases with liver dysfunction and in cases with coronary heart disease diagnosed on clinical and/or electrocardiographic basis.

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BUNDLE BRANCH BLOCK

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INTRODUCTION

Recently, various views have been expressed regarding the relative frequency of right and left bundle branch blocks and the prognosis thereof. Hence, it was thought that a study of various aspects of right and left bundle branch block would be useful.

MATERIAL AND METHOD

Only those cases whose full 12 lead electrocardiograms had been recorded are included, right or left bundle branch block being diagnosed by the unipolar chest leads. There were 45 cases of bundle branch block admitted to J. J. Hospital, Bombay, during the period between the latter part of 1953 and July 1956. They are analysed as regards age and aetiological incidence.

There were 24 cases (53·3 per cent) with right bundle branch block and 21 cases (46·7 per cent) with left bundle branch block. Right bundle branch block and left buidle branch block have occurred with practically equal frequency. Barker (1952) stating the same fact about relative frequency gives the figures of 44 per cent for right bundle branch block and 56 per cent for left bundle branch block.

The age incidence is shown in Table 1.

TABLE 1—SHOWING AGE INCIDENCE

Age group (in years)	No. of cases of bundle branch block		
	Right	Left	Total
0—20 ...	2	0	2
21—40 ...	10	1	11
41—60 ...	6	10	16
61 and above ...	6	10	16

It is seen from Table 1 that while the occurrence of right bundle branch block is fairly distributed at all ages, all cases but one of left bundle branch block fall in the age-group of 41 years and over. The reason for this would be evident while noting the causative factors.

Aetiology—The aetiological factors are shown in Table 2.

TABLE 2—SHOWING ANALYSIS OF AETIOLOGICAL FACTORS

Aetiology	No. of cases of bundle branch block		
	Right	Left	Total
1. Congenital heart disease :			
Atrial septal defect ...	3	0	
Pulmonary stenosis ...	1	0	4
2. Rheumatic heart disease :			
Mitral valve disease ...	3	0	
Aortic valve disease ...	1	0	4
3. Cardiovascular syphilis :			
Aortic regurgitation ...	0	2	2
4. Hypertension	6	7	13
5. Myocardial infarction ...	4	4	8
6. Coronary insufficiency	4	7	11
7. Miscellaneous	2	1	3

Hypertension, myocardial infarction and coronary insufficiency are responsible for 14 out of 24 cases (58·3 per cent) of right bundle branch block, and 18 out of 21 cases (85·5 per cent) of left bundle branch block. As these diseases occur most commonly after the age of 40 years, most of the cases of left bundle branch block have occurred in the age-groups of 41 years and above (Table 1). Below the age of 40 years, congenital heart disease and rheumatic heart disease were aetiologically responsible. All but 3 cases had serious organic heart disease.

Prognosis—10 cases died in the Hospital. Table 3 shows the nature of the heart disease they suffered from.

TABLE 3—SHOWING NATURE OF HEART DISEASE IN THE 10 PATIENTS WHO DIED

Disease	No. of cases of bundle branch block		
	Right	Left	Total
1. Myocardial infarction ...	4	2	6
2. Coronary insufficiency ...	—	1	1
3. Atrial septal defect ...	1	—	1
4. Rheumatic mitral valve disease	1	—	1
5. Syphilitic aortic regurgitation	—	1	1

Prognosis depends on the underlying heart diseases, rather than presence or absence of bundle branch block.

DISCUSSION

Right bundle branch block is considered to offer a more favourable prognosis than the left bundle branch block (Vazifdar, 1952). Papp and Smith (1955) recently emphasised that except in rare instances, it is a sign of organic heart disease.

Some individuals are found to have bundle branch block, but no evidence of heart disease. In the younger age-group (two cases in the present series), the probability is that the conduction defect is a result of myocarditis long since past and completely healed. It represents a static lesion and there is no effect on longevity, cardiac output or reserve capacity.

SUMMARY

45 cases with right and left bundle branch block are studied for their relative frequency, age incidence and aetiological factors.

Right and left bundle branch block have occurred with practically equal frequency.

Aetiologically, coronary artery heart disease and hypertension are responsible for most of the cases, especially those of left bundle branch block.

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SPECIAL ARTICLE

IN VITRO ANTIBODIES

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INTRODUCTION

Antibodies are plasma proteins similar in several of their physicochemical properties to serum globulins but differ in the property to react specifically with their homologous antigens. The organs and tissue cells of immunised animals are capable of producing antibodies *in vitro*. The criteria of production of the antibody, as is well known, is dependent on the fact that the material should be foreign

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to circulation. The antibody response is dependent on this property. Human auto-antibodies in haematologic diseases are reported to erythrocytes, leucocytes and platelets (Dameshek and Schwartz, 1940, Davidson and Spurrior, 1954). Brain tissue cells can produce antibodies to themselves (Rivers *et al*, 1933 ; Rivers and Scwentker, 1935 ; Wakesman and Morrison, 1955). Auto-antibodies reacting *in vitro* with extracts of human kidney have been found in the serum of patients with glomerulonephritis (Lange *et al*, 1949 ; Pfeiffer and Bruch, 1952, 1953). Damage to the lens can be caused by the intravenous injection of lens extracts.

Importance of in vitro Antibodies

In recent years much interest is centred around the site and the mechanism of antibody production, especially the *in vitro* production. The *in vitro* method will help us to define the cellular system involved in the synthesis of antibodies in comparison to the animal as a whole with its complexities. It can also throw light on the difference between the process involving the production of antibody globulin from normal serum globulin, at the molecular level. The importance of this approach could be evaluated from the fact that there was recently an editorial annotation in the *Lancet* (1958).

Methods of in vitro Antibody Production

Tissue cells from lymph nodes and spleen are capable of producing antibodies. In view of this, cells of this type are used as the mediators of antibody production *in vitro*. Primarily two approaches have been used in getting these globulins which are antibodies :

A. Production without the mediation of animal cells. B. Production with mediation of cells.

Production of antibodies without the mediation of cells :

As early as 1940 Pauling reviewed the problem of antibody formation from a physiochemical basis. He suggested that if serum globulin molecules were allowed to unfold by denaturation and later allowed to refold themselves to the configuration of the antigen, the resulting molecule will be antibody. This was based on the facts that antibodies are globulins similar in some of the physicochemical properties but differing from each other in the specificity inherent in antibody from the antigen. Pauling and Cambell (1942) carried out experiments to test this hypothesis.

He exposed bovine gamma-globulin to 56°C for fourteen days in the presence of an azo-dye, an arsonate. This denatured the globulin partly and a precipitate of the dye and protein was formed. This was presumed to consist of a modified globulin united specifically with the "antigen-dye". This dye was then displaced from the precipitate complex by dialysis against a solution of its "determinant group", an arsonate, which was later removed by dialysis against sodium chloride. About 15 per cent of the protein in the resulting solution had certain properties of antibodies. It was specifically precipitated by the azo-dye and precipitation was specifically inhibited by the arsonate. Haurowitz *et al* (1946) repeated this experiment but could not reproduce the results. Boyd (1956) in this connection states that the claim of Pauling and Cambell (1942) has been refuted by various workers besides Haurowitz.

Production of antibodies with the mediation of cells :

Two types of cells have been used in experiments of this kind : 1. Cells from non-sensitised animals. 2. Cells from sensitised donors.

1. *Cells from non-sensitised donors*—These cells from normal animals are brought in contact with the antigen and later made to produce specific antibodies in either of the following ways : (*a*) Culturing these cells which have been in contact with the antigen, by tissue culture methods. (*b*) Injecting these cells into animals which have been made incapable of producing antibodies themselves.

Tissue culture of cells after contact with antigens—The earliest report of antibody formation in tissue culture by the isolated animal cells in the presence of an antigen dates back to 1912, when Carrel and Ingebrigsten demonstrated haemolysins to goat erythrocytes produced by fragments of bone marrow and lymph nodes of guinea-pigs.

This approach was not popular in the period following these reports due to failure to trace the antibodies in experiments done by Salle and McOmie (1937), Kuczynski, Tenenbaum and Werthemann (1925), Sato quoted by Selmar (1944). The failures may be due to certain minor details in the performance of the experiments. The possible explanation to the failure by Salle and McOmie (1937) may be due to addition of heterologous serum to the proliferating culture from the spleen of a rabbit or guinea-pig. In this they were not able to detect the precipitate which could have been very small to be detected with the methods adopted by them and were available then. The other possible explanation is that the splenic and cardiac tissues used for culture and production of antibodies were from chicken embryos. With the present knowledge, it is doubtful whether embryonic tissue is able to perform this task.

Interest has been revived recently in this approach, due mostly to the refined techniques for detection of the antibodies *in vitro*. In recent times there have been three reports of successful production of antibodies *in vitro* by the cells cultured by tissue culture technique after their exposure to the antigens. The first report is by Steiner and Anker (1956). The second is by Stevens and McKenna (1957a, 1957b) and the third by Fishmann (1959).

Steiner and Anker (1956) used individual or clumps of rabbit spleen and bovine gamma-globulin served as antigen. The second group used gamma-globulin and serum albumin of bovine origin, with the cells from the rabbit as usual. They used either Trowell's protein-free culture medium or Earle's medium. Their experiment is given in detail for information.

A normal rabbit was given an injection of 10μg. of purified lipo-polysaccharide from *Salmonella typhosa* by intravenous route. Twenty-four hours later the rabbit was bled and the spleen removed. This spleen so collected was diced and incubated for one hour at 37°C. in Trowell's medium. During this incubation period the medium contained bovine gamma-globulin (BGG) as the test antigen and casein as a control material in varying amounts. Following incubation, the tissue pieces were washed five times in 10 ml. of cold Hank's balanced salt solution without bicarbonate and collected by centrifugation at 700 *r.p.m.* for five minutes at 5°C. Tissues were then planted on tantalum gauze in the same medium as used for incubation but without the antigen.

After varying intervals at 37°C. the tissues were removed from the gauze and homogenised with TenBroeck homogeniser in nine volumes of cold saline. These extracts were centrifuged at 3000 to 5000 *r.p.m.* for 30 minutes at 1-5°C. The supernatant were then titrated for antibody ; these were designated "extracts". The fluids under the tantalum gauze were harvested and titrated for antibody too ; these fluids were designated "fluids" in their reports.

Antibody was estimated by the sensitive Boyden haemagglutination technique as modified by McKenna and Stevens (1958). The *fluid* and *extract* values after incubation with casein were in the non-specific range, so were the values when no antigenic material was added. In contrast, when BGG was present during the one hour incubation, haemagglutination titres averaged eight-fold higher in the fluids and four-folds in the extracts. There was a progressive fall in the fluid titres over three days although the differences were not significant.

Administration of 10μg. of the polysaccharide from the endotoxin from *Salmonella typhosa*, intravenously 24 hours before the BGG contact with the spleen cells increased the antibody response considerably.

At this point a question poses itself. Can the bacterial antigens induce antibody response in a similar condition ? The only answer is that no reports have been seen but it may be possible in view of some other recent reports appearing, where the procedures are a little different, so are the cells used. The reports are with the cells which have been treated in the same way and transferred to the animals in one instance and another one where the bacterial viruses have been used as antigens. These animals who are recipients of these cells are made incapable of making antibodies themselves by irradiation. After the transfer of such cells, antibodies for the specific antigens have been detected, after a period of three to seven days. The peak of titre is best between four to six days. Reports are available for both whole bacteria, bacterial products and bacterial viruses.

In a recent report Fishmann (1959) describes the *in vitro* initiation and production of two different antibodies in cultivated lymph node cells of normal rats. One of the reacting antigens is bacteriophage T_2 and the second one is chemical haemocyanin. In this work, in order to induce the formation of the T_2 antibody it was necessary first to expose the virus to the cells of a rat peritoneal exudate obtained 48 hours after intra-peritoneal injection of beef infusion broth according to the technique by Sawyer *et al* (1954). These cells, mostly macrophages, were washed once in buffered saline and then packed by centrifugation. These cells (approximately $1{\cdot}4 \times 10^6$) were resuspended in 5 ml. buffered saline containing 10^9 T_2 phage. The mixture was incubated at 37°C. for 30 minutes. The cells were sedimented by centrifugation at 1000 *r.p.m.* ; washed twice with buffered saline, ground in a TenBroeck homogeniser and passed through a Seitz filter.

Two millilitres of this cell-free filtrate was distributed among eight Esmarch dishes which contained rat lymph node cells maintained in Trowell's (1955) synthetic medium. Preparations of unheated bacteriophage and of suspension of normal rat macrophages alone, were added to other lymph node cultures. The plates were incubated at 37°C under four per cent carbon dioxide atmosphere. The supernatant fluids from these cultures were removed after varying periods of time and to one volume of fluid was added one volume of saturated ammonium sulphate. The resulting precipitate was disolved in 1/8 the original volume and dialysed overnight against buffered saline of pH 7·6. These concentrated fluids were tested for phage neutralisation by adding dilutions of the

fluid to constant number of T_2 phage, incubating for 30 minutes at 37°C, plating on *E. coli* and counting plaques after 24 hours.

Phage neutralisation was found only with fluids from lymph-node cells that had been exposed to macrophage treated phage particles. The neutralising substance appeared on the fifth day, after which the cells began to lose viability. The pretreatment of the virus by rat macrophages could not be replaced by rabbit macrophages or with HeLa cells. It was also found that rat macrophages which had been damaged by freezing and thawing or by exposure to the Mickle vibrator was ineffective. However, a homogenate from rat macrophages prepared by the use of TenBroeck grinder could be substituted successfully for intact macrophages.

Transfer of cells into non-sensitised animals—Trnka (1958) studied the possibility of antibody formation by the isolated cells after its direct contact with an antigen *in vitro* and tested its capacity to produce antibodies when they were injected in an animal which itself is incapable of producing antibodies. In his experiments he took spleen cells from adult leghorn hens and incubated them in a saline suspension of heat inactivated *S. paratyphi B* or *Brucella suis* for about ten minutes at room temperature.

One milliliter of this suspension was injected into about two-day-old chick of the same species. By using Coombs test he was able to detect antibodies for the organism, produced by the adult mesenchymal cells.

In a later work reported from Czechoslovakia, Miroslav Holub (1958) reported further development to the observations made by Trnka (*loc.cit.*). He used the same antigen, namely *S. paratyphi B* and *Brucella sius* but lymphocytes from cisterna chyli were used. After a direct contact of the antigen, the cells were transferred to a newborn rabbit. Two to four days after the transfer of these 'sensitised' cells the agglutinins were detected.

The works of Harris and Harris (1955) and Harris *et al* (1954, 1955, 1956a, 1956b, 1958a and 1958b), reported in a series of papers on antibody production, are interesting because they not only deal with the problem of *in vitro* antibody production but also elucidate many associated factors that contribute to an increase or decrease in the antibody titre.

In the series of experiments reported in their paper Harris *et al* (1954) have used lymph node cells from rabbit. These cells, after incubation with *Shigella paradysenteriae*, are injected in rabbits to produce antibodies to these bacilli.

Lymph nodes from rabbits which did not receive any injection and also from those animals that received heterologous antigen were collected. These lymph nodes were teased in Tyrode solution without bicarbonate in it but containing gelatin. The purpose of heterologous antigen injection was to stimulate the hyperplasia of the lymph tissue so that the yield of the cells may be high. This does not interfere with the antibody production by the specific antigen that is used. After the lymph cells were collected, they were incubated with a given quantity of dysentery bacilli. After incubation the bacilli were washed away from the surface of the lymphocytes by repeated suspension and centrifugation in Tyrode solution containing rabbit serum. These treated cells were injected into irradiated, but untreated rabbits. Some animals received fresh lymphocytes and the antigen separately.

Agglutinins to dysentery bacilli were found in the sera of the recipient rabbits four days after injection of the lymph node cells which have been incubated *in vitro* with dysentery organism. Agglutinins were also found in the sera of the animals which received the injection of fresh lymphocytes and bacilli separately (without incubation with the bacilli before injection). From this it is presumed that after contact of the antigen with lymphocytes in test tube or in the body of the recipient animal, these cells synthesise the specific antibodies.

These results also indicate no difference between the appearance of agglutinins after the transfer of cells whether these cells had been incubated with the antigen at 37°C for 30 minutes or mixed in an ice-bath temperature before washing.

In their previous work Harris *et al* (1954) had shown that a short interval of ten minutes of contact of the antigen *in vitro* with the animal lymphocytes is sufficient to induce them to produce the antibodies in irradiated recipients after three days, after injection of the cells. The peak of antibody production was reached in four to six days.

It has also been shown in their work that the lymphocytes should be uninjured to be active in producing these antibodies. The report also indicated that both the lympocytes as well as the spleen cells share this capacity of producing agglutinins to the antigens used in the irradiated animals.

In the later work of Harris *et al* (1955) there is evidence that the whole killed bacteria and culture filtrates could induce antibody production by the cells concerned. The bacilli were incubated in the serum and this serum so prepared was used instead of the organisms. The results were similar to those in the earlier instance.

2. *Antibody production by sensitised cells*—Studies by McMaster *et al* (1935) provided experimental evidence of the antibody production in the

lymph nodes of animals receiving the injection of an antigen. It was also shown that spleen cells too shared this capacity (Harris *et al*, 1948). Inspite of this and other studies it is generally admitted that the plasma cells are implicated in antibody formation.

Antibody production has been studied in two ways: (*a*) In tissue culture. (*b*) By injecting sensitised cells into recipient animals.

Antibody production by sensitised cells in tissue culture—A variety of antigens have been employed in these experiments. Antigens range from erythrocytes (Robert *et al*, 1949), *Paratyphoid B vaccine* (Thorbecke and Keuning, 1953), formalin-killed type VIII pneumococci (Ranney and London, 1951), *Salmonella typhi* (Fagraeus, 1948) bovine gamma-globulin (Robert and Dixon, 1955) and diphtheria toxoid (Stavitsky, 1957).

The most popular of these antigens is diphtheria toxoid in view of the following advantages over others as reported by Stavitsky (1957): (i) Natural antibodies (antitoxoids) are not found in normal non-sensitised experimental animals. (ii) The diphtheria antitoxin can be determined quantitatively. (iii) A second injection of diphtheria toxoid yields more antibody per milligram of antigen than others.

Stavitsky (1955) has shown that lymph nodes and spleen fragments from immunised animals can produce diphtheria antitoxin when these fragments are placed in culture tubes and rotated at 37°C. It has been shown that the quantity of *in vitro* antibody synthesis is the same as *in vivo*.

Antibody formation by sensitised cells after transfer—One of the fruitful approaches in the study of antibody production is the transfer of cells and tissues of an immunised donor to a non-immunised recipient. This has disclosed several finer points regarding the possible sites and mechanism of antibody production. This cannot be regarded as an *in vitro* system in the technical sense of the word because the antibodies are found in the tissues of the recipient and hence they are *in vivo*. It has been argued that the tissues of recipients themselves may not take part in synthesis actively because the animals used as recipients are irradiated and made incapable of immunological response. It has been shown that they are incapable of *in vivo* production to any antibody. The objection raised at this point and the argument put forward in favour of the claim of calling it conveniently *in vitro* holds true in an earlier instance too. This was in connection with the transfer of cells from non-sensitised animals but brought in contact with the antigen and then transferred.

Effect of Metabolic Inhibitors

Respiratory inhibitors such as dinitrophenol, cyanide and such other substances inhibit antibody production, indicating the energy requirement for the process (Dutton *et al*, 1958). The purine analogues such as 8-azo-guanine has similar deleterious effect. This shows that the net synthesis of ribonucleic acid is needed for antibody production (Creaser, 1956). Purine analogues are known to inhibit the induced enzyme synthesis in bacteria. This study indicates a close similarity to the induced enzyme synthesis in bacteria. There is considerable evidence to indicate that the mechanism of antibody production *in vitro* may be very similar to that suggested for the synthesis of induced enzymes (Schweet and Owen, 1957).

Antibody Production by Single Cell

Nossal and Lederberg (1958) using the micro-technique have isolated single cells from the lymph nodes of animals which were simultaneously stimulated with two antigens. The isolated cells formed antibodies specific to one or the other antigen used.

White (1958) in his recent paper has reported some more aspects of cellular antibody production *in vitro*. He proposed a hypothesis that these cells containing a particular antibody group themselves into a clone. It was possible to study this clonal distribution of the cells by fluorescent antibody technique of Coons.

Lennox *et al* (1959) present a hypothesis different from that of Nossal and Lederberg (1958). They report that in their experiments there is evidence of multiple antibodies by an individual cell. This report is interesting from other points that are raised in it. They developed a method to assay the antibody released by the lymphoid cells from the rabbit which had received injection of two serologically unrelated bacteriophages T_1 and T_2. It appears that 10 to 15 per cent of these cells appear to be involved in the synthesis of antibody inactivating these phages. Their evidence is that isolated cells are actually synthesising rather than releasing stored antibodies made elsewhere. About 10 per cent of these cells that are actively producing antibody are of lymphoid series and 30 per cent are of plasmocytic series. These cells isolated from animals immunised with both phages make a measureable amount of either T_1 or T_2 and some make both. Control experiments favour the assumption that a cell showing double activity is actually producing both the antibodies.

Precursors of *in vitro* Antibodies

Problem of precursors to antibodies and their origin has been attracting attention these days.

The precursors of antibodies could be either amino acids in culture medium foming the pool or some preformed polypeptides. There is evidence for both the possibilities, but the final word about it could be sought by further research. Green and Anker (1958) and later Stavitsky (1958) using isotope technique have shown that the precursors are amino acids found in the culture medium.

According to the hypothesis proposed by Karush (1958), the polypeptides are the precursors. The disulphide bonds of the antibody molecule formed from these polypeptides play a key role. The polypeptide chain after its formation according to template, spontaneously forms itself into gamma-globulin precursor with a sulphydryl group and forms a complex. During the existance of the complex so formed, oxidation of the sulphydryl group occurs and leads to the stabilisation of specific sites of antibody. This stage of reaction is reversible. The antigenic group so formed and released reacts with fresh batch of gamma-globulin precursors with the reduced disulphide bond. In this way a cyclic reaction is set. Lederberg (1959) makes an amendment to Karush's hypothesis. He thinks that determinant amino acid sequences may also be involved besides the disulphide bond and the sulphydryl group. Further study will help one to find the correct picture.

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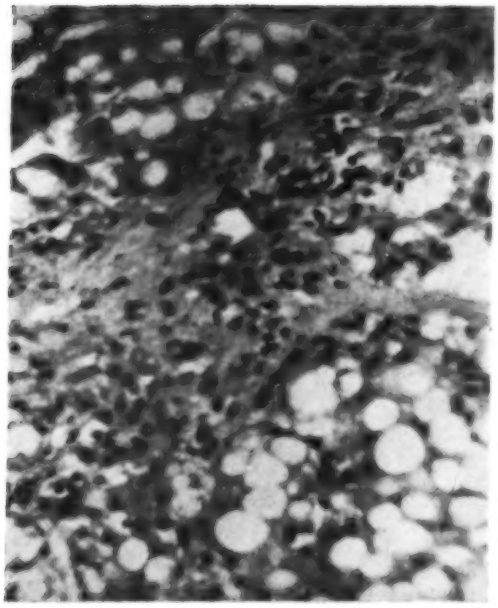

Fig. 1—Infantile cirrhosis showing fibrous tissue proliferation and fatty metamorphosis with numerous fat cysts. This was seen in the case whose age was 5 years. This change is uncommon in infantile cirrhosis and is seen in children of higher age group. (H. and E. × 200)

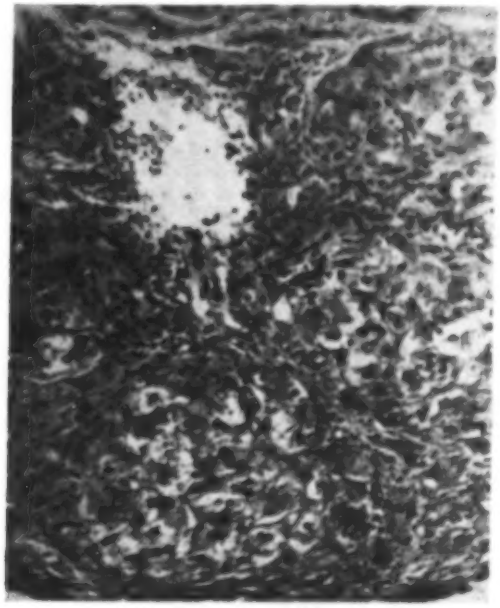

Fig. 2—Infantile cirrhosis from a well established case showing pseudolobulation, regenerating cells in the pseudolobules and inflammatory cells in the peripheral area. (H. and E. × 200)

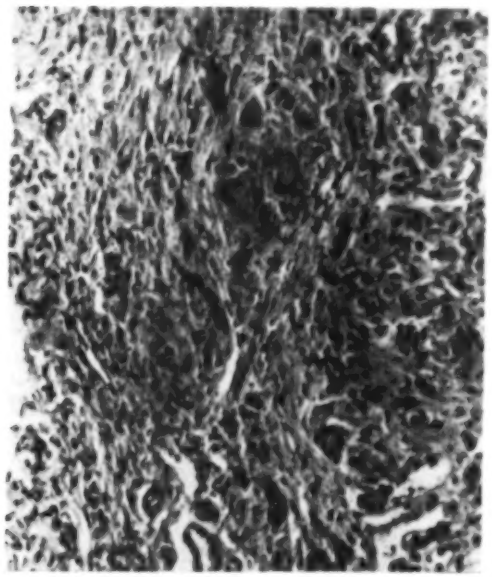

Fig. 3—Infantile cirrhosis from an advanced case showing absence of lobular pattern, extensive fibrosis which separates small groups of cells or even single cells. (H. and E. × 130)

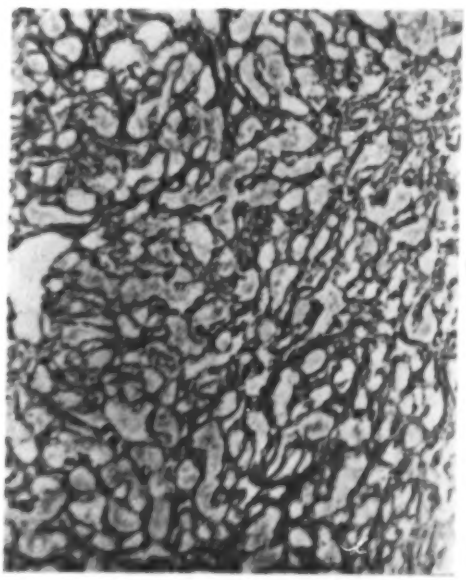

Fig. 4—Infantile cirrhosis from an advanced case showing the disposition of reticulin fibres. The absence of lobular pattern is noteworthy. (H. and E × 130)

BERRY AND SAINANI—*Infantile Cirrhosis (p. 121)*

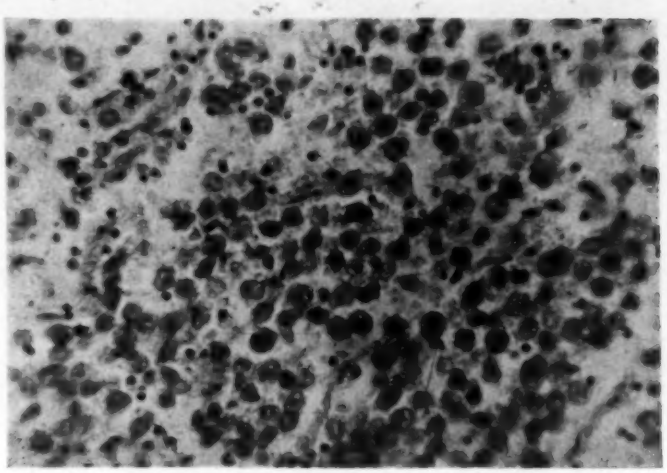

Fig. 1—Shows masses of round cells in alveolar arrangement ; the cellular and alveolar arrangement is anaplastic ; the cytoplasm is mostly disintegrated making the nuclei and nucleoli prominent (Case 1).

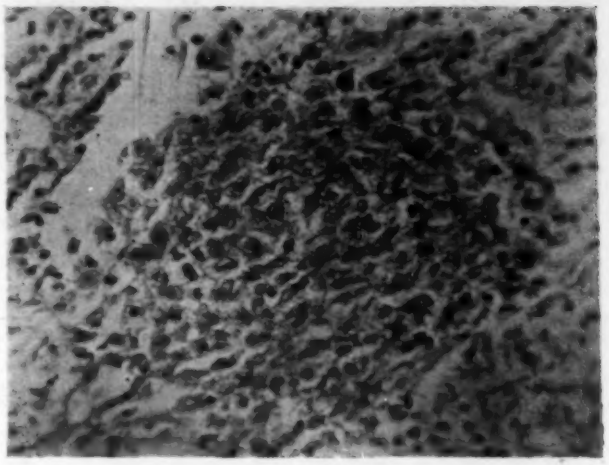

Fig. 2—Shows the tumour cells as fairly large with cytoplasm pale, margins indefinite and nuclei vesicular with one or two nucleoli and areas of necrosis and haemorrhage at places (Case 2).

LAHIRI AND ROY—*Malignant Abdominal Testis (p. 139)*

Fig. 1—Showing ptosis before prostigmine injection (Case 1).

Fig. 2—Showing improvement in ptosis (same case) 15 minutes after 1 mg. prostigmine injection.

Fig. 3—Showing improvement in ptosis (same case) 30 minutes after the injection.

JAIN AND AGARWAL—*Myasthenia Gravis (p. 141)*

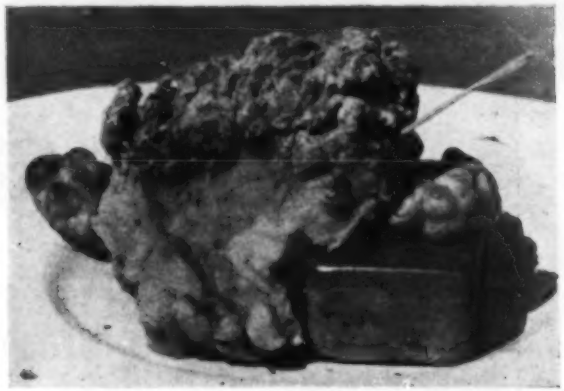

Fig. 1—Showing the specimen of the uterus removed with appendages, two cystic ovaries and the growth, the pointer indicating invasion deep into the muscle.

CHOUDHURI—*Chorionepithelioma of the Uterus (p. 142)*

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CASE NOTES

MALIGNANT ABDOMINAL TESTIS

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The imperfectly descended testis is liable to all the diseases which can affect the fully descended organ. The retained testis is peculiarly prone to some of these affections, malignant change being one of them. Indirect inguinal hernia is a concomitant finding in the majority of cases, almost always this is present with retained testis (Gross, 1953). It is impossible to determine whether malignancy occurring in an undescended testis is due to malposition or is a reflection of congenitally defective structure which is also responsible for failure of descent. The possibility of congenital or constitutional inferiority of the testis appears to be a more likely explanation (Sohval, 1954; Whittle, 1957) for tumours have developed even after orchidopexy and also following late spontaneous decent (Gilbert and Hamilton, 1940; Scott, 1954-55; Som, personal communication).

In an exhaustive survey of malignant testicular tumours, Gilbert and Hamilton (1940) found 11 per cent in cryptorchids. Since the incidence of retained testis in adult population has been estimated to be in the neighbourhood of 0·2 per cent (Alyea, 1929; Gilbert and Hamilton, 1940), it appears that malignancy is approximately fifty times commoner in the undescended than in the normal testis (Gilbert and Hamilton, 1940; Sohval, 1954). Gilbert and Hamilton (*loc. cit.*), from their analysis of the reported cases, found that the tumours of the abdominal testis were four times commoner than those of the inguinal testis. According to these figures, one in every 20 abdominal testes as compared to one in every 80 inguinal testes, might be expected to undergo malignant change (Gilbert and Hamilton, 1940; Dixon and Moore, 1952).

It is of interest to note that cases of malignancy in testis after orchidopexy, have been reported in literature, the tumour becoming evident approximately ten years later (Gilbert and Hamilton, 1940; Whittle, 1957).

Malignancy of abdominal testis is a rare condition and the purpose of this communication is to record two such cases.

CASE REPORTS

CASE 1—A man, aged 32 years, was admitted to the hospital on 3-1-56 on account of a small painless swelling of three months' duration over the suprapubic region. It gradually increased in size and at the same time his general health was deteriorating. A mass of about 3" in diameter, over the suprapubic region a little towards the right side, was detected on clinical examination. It was firm and slightly tender ; the margin of the swelling was indefinite and the local temperature was normal. Absence of the right testicle in the scrotum was the only abnormality detected.

Investigations—R.B.C. count was 3·2 million/cmm. Other laboratory findings were normal.

The skiagram of the chest was normal.

Operation—On 8-1-56 operation was performed through the right lower paramedian incision. A mass, about 4" × 3", was found in relation to the spermatic cord. The macroscopic appearance was very suggestive of testicular tumour. It was not adherent to any other organ or tissue of the pelvis ; and the lymph nodes were not enlarged. After removing the tumour, the abdomen was closed. He made an uneventful recovery, and 10 days after the operation he was discharged from the hospital. Unfortunately he did not return to the follow-up clinic for further advice and radiotherapy.

Histology of the specimen—"The section shows masses of round cells in alveolar arrangement. The cellular and alveolar arrangement is anaplastic. The cytoplasm is for the most part disintegrated, and as a result the nuclei and the nucleoli only are seen. The diagnosis of the tumour is seminoma" (Fig. 1, *vide* Plate).

CASE 2—A man, aged 27 years, was admitted to the hospital on 5-2-58 with a painful lump on the left side of the lower abdomen. Two months before admission, while at work he felt a jerk in his lower abdomen followed by pain and a few days later discovered a lump on the left side of the lower abdomen. It gradually increased in size and became painful. He had occasional low-grade fever.

Clinical examination revealed an intra-abdominal oval lump about midway between the umbilicus and pubis, slightly to the left side. Local skin temperature was raised. There was a left indirect inguinal hernia. The left testis was absent in the scrotum. The right organ was in normal position, and the development of the scrotum was normal.

Investigations—Laboratory examinations of blood, urine and stool revealed nothing abnormal.

The skiagram of the chest was normal.

Operation—On 12-2-58 the left inguinal hernia was repaired through an inguinal incision, and then a sub-umbilical midline incision was made for exploration of the lump. On opening the abdomen a fungating mass was discovered in the left iliac fossa ; the mass was diagnosed to be the left abdominal testis which had undergone malignant changes. There was much adhesion among the coils of the intestines, particularly in the

pelvic colon. The tumour was removed as thoroughly as possible and the abdomen closed. The aortic lymph nodes were not involved. Apart from slight pyrexia, the patient made an uninterrupted recovery, and was discharged from the hospital 12 days after the operation. He was advised a course of post-operative radiotherapy.

Histology of the specimen—"The section shows large sheets of tumour cells separated by fibrous septa. The cells are fairly large with pale cytoplasm having indefinite margins. The nuclei are vesicular with one or two prominent nucleoli. Areas of haemorrhage and necrosis are also visible here and there. The tumour is a seminoma". (Fig. 2, *vide* Plate).

Discussion

Tumour of the testis, irrespective of its situation, is a rare condition. Malignancy in abdominal testis is still rare, since the incidence of cryptorchism is 0·1 to 0·2 per cent in all young adults (Alyea, 1929).

The two cases reported proved to be seminoma on histological examination although teratoma was found to be the commonest new growth of the undescended testis.

An abdominal testis is usually not palpable unless it is pathological. A palpable abdominal testis should be diagnosed clinically as malignant until proved otherwise. The Aschheim-Zondek reaction has fallen into disuse as far as diagnosis and the detection of metastasis are concerned (Whittle, 1957). Whittle (*loc. cit.*) recorded that this test was performed on 34 occasions but in only six cases was it positive. This test was not performed in either of the cases reported here.

The treatment of the condition is the same as in malignant tumour of the scrotal testis; orchidectomy and post-operative irradiation to the retroperitoneal lymph nodes are generally advised in every case. When dealing with the malignant abdominal testis surgically, exploratory laparotomy is a better procedure than removal of the tumour through the inguinal incision as it helps the surgeon to assess the extent of the lymphatic spread and also involvement of other organs by the malignant growth. A separate inguinal incision may be required for an associated hernia, as has been made in Case 2. Result of treatment of this unfortunate condition is far from satisfactory, although Whittle (*loc. cit.*) reported two such cases who survived a period of ten and fifteen years respectively.

The important fact worth emphasising is that a testis which remains in the abdomen and becomes malignant has little chance of being discovered until tumour extension or metastasis has occurred whereas malignancy developing in a testis, which has been brought down into the scrotum or was in the scrotum, has the possibility of early detection. From the point of view of possible subsequent malignancy, the management of the undescended testis in the adult patient has been the subject of considerable concern and controversy. Conceding the incidence of testicular tumour to be very low (0·0013 per cent for living males) nevertheless it appears to occur about fifty times as often in cryptorchids as in men with scrotal testes (Gilbert and Hamilton, 1940; Sohval, 1954). Despite these observations, it is the general opinion that the incidence is still too low to justify prophylactic orchidectomy, and also that probability of malignancy should not be the reason for advising orchidectomy. However, since an intra-abdominal testis, as compared to an inguinal one, is several times more prone to malignant transformation (Dixon and Moore, 1952; Gilbert and Hamilton, 1940), an abdominal retained testis should be viewed with considerable concern. When, however, surgery is performed for an associated symptomatic hernia or for psychological reasons, the decision, in cases of testes which are seemingly impossible to bring down in the scrotum, may be difficult. The balanced approach to the problem would be that if an abdominal testis cannot be placed in a position where it can be felt, orchidectomy should be performed provided the condition is unilateral. The inguinal testis should never be placed in the abdomen if it cannot be brought into the scrotum. The rationale of orchidopexy must rest on the placement of the testis where it can be observed closely. Undescended testes, which cannot be brought down satisfactorily at the time of surgery, are not to be subjected to multiple stage operations. They are best left alone if in the groin, and removed if in the abdomen (Grove, quoted by Scott, 1954-55). Surgical judgment is guided by proper evaluation of the benefits to be obtained from hormone secretion of the testis and the danger of carcinogenesis; proper spermatogenesis can hardly be expected in an abdominal testis. It is worth recalling that parenthood is not denied in all cases of bilateral undescended testes; a 65 year old man with bilateral abnormality is known to be the father of 7 children (Som, personal communication).

Summary

Two cases of malignant abdominal testis have been described. Both patients were adults and came with a lump in the lower part of the abdomen; the signs of tenderness and local rise of temperature in one case were due to fungation of the tumour whereas these signs were absent in the other reported case.

When a palpable abdominal mass is the presenting clinical feature of a cryptorchid the possibility of malignant change has been stressed.

Important surgical problems are discussed.

Acknowledgments

We are indebted to Dr. A. K. Dutta Gupta, the Superintendent, N. R. Sircar Medical College Hospital, for permission to use the hospital records, and to Prof. A. L. Som for his generous assistance and helpful criticism. We thank Prof. K. N. Basumallik for the microphotographs and the pathological reports.

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MYASTHENIA GRAVIS

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The clinical features of the malady for which the name myasthenia gravis pseudoparalytica was first given by Jolly in 1895, form a peculiar syndrome characterised by weakness of the voluntary muscles, especially those supplied by the cranial nerves. As a result ocular movements, facial expression, mastication, deglutition and speech are often affected early. Cardiac and smooth muscles are not affected. There is thus a resemblance to bulbar or bulbopontine palsies on the one hand and to muscular dystrophies on the other. The affection is sometimes fatal, often serious, liable to remissions and relapses, and spontaneous recovery may occur.

Historical review—Although myasthenia gravis was not described as a clinical entity with any completeness, until the last quarter of the 19th century, a reference by Thomas Willis (17th century) indicates that he knew the disease and recognised the chief symptoms of asthenia of the voluntary muscles, with recovery after rest.

The syndrome was then referred again after 200 years by Wilks (1877) who, in describing some cases of bulbar paralysis and other neurological syndromes, described one patient with symptoms slightly suggestive of those seen in case of myasthenia gravis.

A more important presentation was that by Erb (1879), who may be regarded as the first physician to write a reasonably full account of the syndrome. Erb's paper though incomplete, is of value, however, for Erb first brought out clearly some of the chief symptoms of myasthenia gravis, particularly the distribution of the paralysis in the muscles supplied by the bulbar nuclei, the progress by remission and relapse, the sudden death, and the frequency of ptosis as the leading sign of the disease.

Hoppe (1892) reported a fully studied case with no abnormal findings at the postmortem examination.

Goldflam (1893) added greatly to our knowledge of the disease. To differentiate his cases from those of bulbar paralysis he pointed out : the lack of atrophy of the muscles affected, the absence of fibrillation, the normal electric responses, the presence of sphincter control, the normal or lively reflexes with reflex exhaustion after continuous stimulation and the normal palatal reflex.

Jolly (1895) first gave the name myasthenia gravis pseudoparalytica to the disease. He was the first to analyse the reaction of the muscles in myasthenia gravis to electrical stimulation. He also suggested physostigmine as a form of treatment but he did not follow out this point and it was left to Walter to demonstrate the therapeutic value of this drug.

Murri (1896) reported a case under the name of Erb's disease. He used electrical reactions and pointed out that even when complete exhaustion was produced by an electric current the muscles could readily contract voluntarily. He also showed the reverse that muscles exhausted voluntarily would still react to the electric current.

Karplus (1897), quoted by Viets (1953), separated from the general group of asthenic paralysis those with ophthalmoplegia as the first and most persistent symptom and called them asthenic ophthalmoplegia.

CASE REPORTS

CASE 1—J. K., a farmer, aged 24 years, admitted on 10-6-59, complained of drooping of both the eyelids for 3 months. He was compelled to move his head to look sideways and had difficulty in mastication. There was no history of diplopia at any time nor any appreciable improvement after rest.

On examination—There was ptosis of both the eyes in an expressionless face. The masseters were both equally weak. There was generalised weakness of the muscles supplied by the facial nerve. The nasolabial folds were less prominent.

Ocular examination—Bilateral ptosis was present. The patient was unable to use his frontalis muscle as well, to compensate for the ptosis. All the ocular movements were restricted to such an extent as to give a picture of complete external ophthalmoplegia. The pupils were reacting briskly to light (direct and consensual). The vision in both the eyes was 6/6.

Ophthalmoscopy revealed no abnormality.

The examination of the other cranial nerves and of the motor and sensory nerves and reflexes showed no abnormality.

Investigation—The B.M.R. was −5.

Prostigmine test—On giving an intramuscular injection of 1 mg. of prostigmine, the patient showed improvement in 15 minutes and was completely free of symptoms in 30 minutes (Figs. 1, 2 and 3, *vide* Plate).

Routine examination of blood for total and differential counts, Kahn's test, urine examination and skiagraphy of the chest did not reveal any thing abnormal.

CASE 2—P., a student, aged 19 years, was admitted on 22-4-1958 with the complaints of drooping of both eyelids, difficulty in moving his eyes so that he had to move his head. He also complained of burning sensation in the eyes and headache on reading. Duration—5 years.

On enquiry the patient said that there was marked improvement after rest. There was no history of diplopia at any time or any other complaints or feeling of weakness anywhere, on activity.

On examination—There was ptosis of both the eyes, more of the left eye.

Ocular examination—All the ocular movements were restricted, more so in the left eye giving a picture of partial ophthalmoplegia externa. Pupils were reacting to light and accommodation. Vision in both the eyes was 6/6.

Clinical examination of the other systems and laboratory investigations as in Case 1 proved negative.

Prostigmine test—On giving an intramuscular injection of 1 mg. of prostigmine the patient showed improvement in 10 minutes and was completely free of the symptoms in 20 minutes.

DISCUSSION

Both the cases presented with ptosis and external ophthalmoplegia. Ptosis, which is considered as a leading and most common sign, was first pointed out by Erb as early as 1879.

The first case had external ophthalmoplegia along with involvement of the muscles of mastication of three months' duration; while the second case had only external ophthalmoplegia for period of five years. Karplus (1907) separated from the general group, the cases with ophthalmoplegia as the first and persistent symptom and designated them as asthenic ophthalmoplegia.

When localised ocular myasthenia gravis becomes generalised, it usually does so within the first year of the disease. The majority of patients who suffered from localised ocular myasthenia for more than a year did not develop further extension of the disease during a period of observation of 1-41 years (average 8·5 years). (Case 2 had localised ocular myasthenia of 5 years' duration).

Weakness in relation to activity is supposed to be the keynote in diagnosis. Case 1 did not show either weakness on performing work or any appreciable improvement after rest. Cecil and Lobe (1952) mention that muscles involved have a continually reduced function which has been found in many cases. Evidence of enlargement of the thymus was absent in both the cases. According to Grob (1953), thymoma or thymus hyperplasia occurs in from 10-40 per cent of cases. So far no conclusive evidence of relationship to the thymus has been established.

Diplopia, another important symptom, was very conspicuously absent in both the cases.

SUMMARY

Two cases of myasthenia gravis have been reported. Both had external ophthalmoplegia as the first and persisting symptom.

ACKNOWLEDGMENT

Our thanks are due to Dr. R. Bhattacharya, Superintendent, M. Y. Hospital, Indore, for permission to report the cases.

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CHORIONEPITHELIOMA OF THE UTERUS FOLLOWING AN INCOMPLETE ABORTION

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INTRODUCTION

Chorionepithelioma is the most malignant neoplasm arising from the embryonic chorion, the trophoblasts. The commonest site of its occurrence is the uterus. It occurs more commonly after vesicular mole than after an abortion or after termination of pregnancy at term. An important contribution has been made to our knowledge by Novak and Seah (1954) who studied materials from 72 cases collected by the Mathieu Memorial Chorionepithelioma Committee of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons. Possibly this is the largest group of cases in the world literature that has been studied by a single group of pathologists. Chorionepithelioma generally occurs within a few weeks to a few months from the evacuation of the vesicular mole or after an abortion or after the termination of pregnancy at term. Very occasionally it happens long after pregnancy.

If one accepts Novak's estimate that vesicular mole occurs in 1 in 2,500 pregnancies and that 1 to 2 per cent of vesicular moles changes into chorionepithelioma then it can be assumed that the incidence of chorionepithelioma is 1 in 50,000 to 100,000 pregnancies. As will be shown later 40 per cent of chorionepithelioma followed vesicular mole. According to Novak and Seah (*loc. cit.*) 39·2 per cent followed vesicular mole, 37·8 per cent followed abortion and 23 per cent followed preg-

nancy at term. Hasegawa from Japan, on the other hand, found mole in 67·9 per cent, abortion 25·9 per cent, pregnancy at term 6·3 per cent.

In our department from March 1958 to July 1959, we had 7 cases of vesicular mole and one case of chorionepithelioma following abortion. None of our cases of vesicular mole developed chorionepithelioma uptil now.

Considering the rarity and virulence of the disease, a case of chorionepithelioma following an incomplete abortion is being presented. It will not be out of place to mention that majority of the patients die within six months and few survive more than two years. We were handicaped in this case not having any privilege of doing a pregnancy test.

Case Report

S. B. D., aged 33 years, a very ill patient was admitted on 8-9-58. She stated that sometime in the middle of August 1958 she passed a foetus per vaginum preceded and followed by severe bleeding. She was not sure whether she passed the placenta along with it. She had a period of amenorrhoea of 4 months. Since then she was bleeding off and on, which made her weak. She was para 6+0. All the previous labours were uneventful and the babies were alive.

On examination she was found very pale. The pulse was of low volume and tension with a rate of 140 per minute, the respiration rate was 24 per minute; the temperature was 100°F. Haemic murmur was detected in all the areas of the heart.

The vaginal outlet and the vagina were pale. The cervix was eroded; the external os was closed. The uterus was of the size of 10 weeks pregnancy. The appendages were tender but there was no mass. The ovaries were palpable and normal in size. She was not bleeding at that moment.

Provisional diagnosis was incomplete abortion.

Laboratory investigation—Haemoglobin was 30 per cent and there was slight leucocytosis.

Management—She was left with sedatives, antibiotics and haematinics. 560 c.c. of blood was transfused 3 days after her admission. With another 280 c.c. of blood she was put up for surgical evacuation of the uterus.

By gentle dilatation, the uterus was evacuated. During evacuation some difficulty was encountered. At one place the placenta was densely adherent to the uterine wall. The evacuation was probably complete. After evacuation the bloodstained discharge stopped within 48 hours. She was kept in the hospital for 15 days more because of her general debility and anaemia. She was then discharged with proper advice.

Readmission—On 17-12-58, three months from her first admission, she was re-admitted in our unit. She started her first vaginal bleeding at home after the operation for evacuation, on 6-12-58 and since then she was having prolonged and rather heavy bleeding.

Pelvic examination: Outlet—relaxed; cervix—bluish in colour, closed, very soft. Uterus—size of 10 weeks' pregnancy, very soft. No internal ballotment, Hegar's sign was positive. Two doubtful cystic masses were detected in both appendages. She was bleeding in moderate amount during the examination. On repeated questioning both the patient and her husband denied any history of sexual intercourse during this period.

Provisional diagnosis was chorionepithelioma.

Operation—Just to confirm the diagnosis clinically, two laminaria tents were put into the cervix on 19-12-58, the day before that scheduled for operation. On the operating table the tents were taken out. The finger was pushed through the cervix and it was found that the uterus was filled with fleshy masses. The diagnosis was thus confirmed. Transfusion of 280 c.c. of blood was given and a total abdominal hysterectomy with bilateral salpingo-oophorectomy was performed. Before the abdomen was opened, the external os of the cervix was stitched up vaginally, just to prevent spilling of malignant tissue.

The specimen contained the total uterus with its appendages and the two cystic ovaries. The growth was confined to the cavity of the uterus. It did invade deep into the muscle (Fig. 1, *vide* Plate). The peritoneal surface was smooth. The ovaries contained multiple cysts with bluish fluid material; these were multiple leutin cysts of the ovaries, commonly associated with chorionepithelioma.

The diagnosis was confirmed histologically.

The patient had an uneventful recovery.

Before she went home an x-ray of the chest showed no evidence of metastasis. She improved in general health and put on weight.

Follow-up—She came for follow-up only once in February 1959. She was putting on weight. There was no evidence of clinical or radiological metastasis. In the middle of April 1959 she came to us with intractable headache. She was admitted again in the hospital. A provisional diagnosis of metastasis of chorionepithelioma in the brain was made. X-ray of the skull was suggestive of metastasis but was not confirmatory. The lungs were free. A few days after her admission in another hospital she died, where a diagnosis of widespread metastasis of chorionepithelioma was confirmed.

Discussion

Chorionepithelioma of the uterus is the most malignant disease which affects women at any age of the childbearing period. This disease limits ones longevity

in most cases from between six months and two years. As we are in the dark about the aetiology of chorionepithelioma, it is to be pointed out that charionepithelioma of the uterus is not a disease of the uterus primarily, but of the embryonic chorion. The uterus is secondarily involved. It is unfortunate that the uterus is mixed up with its nomenclature. This disease is a clinicopathological problem. In a case of incomplete abortion or after termination of a normal pregnancy if a patient starts bleeding vaginally, one should first think of bleeding being due to incomplete evacuation of the uterus and then of chorionepithelioma as in the case under review. The clinician always expects, rather depends on, the histologists for the final diagnosis. In a frank case of chorionepithelioma with a growth in the uterus invading the musculature with haemorrhage and necrosis as in the case under report, the diagnosis may not be difficult. Histologically, in the case presented, the proportion of Langhans' cells and syncytium was normal, there were no demonstrable villi. The only sure point to diagnose uptil now that has been put forward by almost all authors is that if the villus pattern is maintained one should lean back from the diagnosis of chorionepithelioma. In Novak and Seah's series in only one out of 74 cases were a few villi demonstrable in a tumour which metastasised and killed the patient.

ACKNOWLEDGMENTS

I am grateful to the C. M. O. H. and D. M. O., Murshidabad, for their permission to handle the case records. I thank Dr. U. C. Chakravarty, Consultant, Mayo Hospital, Calcutta, for his kind help. I express my appreciation to Dr. Miss Maitrayee Ghosh, Lady Assistant Surgeon of our Hospital, and thank Sister Nirupama Banerjee along with her nursing staff for their kind nursing care of the patient.

REFERENCE

NOVAK, E. AND SEAH, C. S.—*Am. J. Obst & Gynec.*, **67**: 933, 1954.

TRANSFUSION SYPHILIS

P. R. PRABHAKAR, L.M.P., CAPT. (EX-I.A.M.C.)

Venereologist, Jammu Province, Jammu, Tawi

CASE REPORT

Mrs. K., aged 35 years, was referred to our clinic by the Medical Outpatients' Department of S. M. G. S., Jammu, on 7-11-58 with fluorescent eruptions all over the body, similar in appearance, character and distribution to those of secondary syphilis. There was no history of venereal sore or extramarital exposure in her or her husband's case.

She was admitted in the gynaecological ward, on 20-8-58 for severe anaemia after delivery. She had septic fever after a difficult labour. Her haemoglobin index at that time was 60 per cent with a R.B.C. count of 2·5 million per c.mm. She was given a transfusion of blood (450 c.c.) on 23-8-58. The blood was from a professional donor.

About 20 days after the transfusion the rash appeared and she was seen when referred to our department about 7 weeks later on 7-11-58.

Laboratory investigation—Her blood was tested on 11-11-58 and 15-11-58 with results absolutely identical on both occasions. Her husband's blood was also taken along with hers and tested on 11-11-58. The donor's blood was also tested and found positive (Table 1).

TABLE 1—SHOWING RESULTS OF SPECIFIC TESTS OF BLOOD OF THE PATIENT, HER HUSBAND AND THE DONOR

	M. K. R.	V. D. R. L.	Kahn
Patient's blood ...	+ + +	Strongly positive 1 in 32	+ + + +
Husband's blood ...	Negative	Negative	Negative
Donor's blood ...	+ + +	Strongly positive 1 in 32	+ + + +

Previous record of S.T.S. was gone into, and it was found that she had her blood tested when admitted into the gynaecological ward on 20-8-58 (3 days before the transfusion) and was found to be negative (M.K.R., V.D.R.L., Kahn).

Treatment—She was given the routine treatment of syphilis (PAM 4·8 mega units, in 4 injections and 20 c.c. of bisoxyl, 10 weekly injections of 2 c.c. each). Her blood was retested on 10-2-59 and again on 25-4-59 and was found negative by all these tests.

DISCUSSION

The following are the reasons for diagnosing the case as one of transfusion syphilis :

1. Her blood was negative on 20-8-58, i.e., 3 days before the transfusion.
2. She was given transfusion of blood on 23-8-58 from a donor, later on proved to be strongly positive for S. T. S.
3. Her blood was found to be positive on two occasions at 4 days' interval when she had fluorescent secondary rashes.
4. Her husband's blood was negative when hers was strongly positive.
5. Her donor was strongly positive.
6. Her blood became negative after one course of anti-syphilitic treatment within 3 months. This quick negativation of S.T.S. is very strongly in favour of the belief, provided by other factors that infection though heavy was fresh.

SUMMARY

A case of syphilis acquired through transfusion of blood from an infected donor is described.

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CALCUTTA, FEBRUARY 16, 1960

THE PHYSICS OF MURMURS

In 1829 Corrigan performed certain experiments and concluded that murmurs are produced by a purely physical cause—the alteration in the motion of blood flow. Fifteen years after a new theory was put forward by Rouanet ; according to him the formation of eddies, vortices or *tourbillons* is the basis for murmur production. He was also of opinion that dilatation of arteries or stenosis of orifices and insufficiencies facilitate murmur production. That the cause of murmur production lies in turbulence has remained an accepted fact since then. Turbulence as the cause of murmur was put on a more scientific basis in 1883 by Reynolds[1], a professor of engineering. He constructed a model and with the help of a dye showed that at a low rate of flow a streamline is maintained and above certain critical velocity, the flow becomes turbulent. The degree of turbulence was directly proportionate to the diameter of the pipe and viscosity of the fluid. He incorporated these findings in a formula for a value known as Reynolds number. When R exceeds 2000, a streamline flow becomes turbulent. This number is known as critical value.

Turbulence, having such a sound background, was accepted as the only cause of murmur and received the full support of physiologists[2], who, while discussing causes of murmur, state that the replacement of a normal streamline flow by a turbulent flow contributes to the production of the abnormal sound.

But the cardiologists seem to have differed. Levine[3] is of opinion that the velocity of blood flow is the only one possible mechanism in the production of a systolic murmur. He suggests that the murmur is produced because of the ejection of blood with a snap. It has been shown that there is a speed with which a fluid running through a tube does so without producing eddies and bruits and beyond which such disturbances do arise. White[4] holds the view that vibration of the valves, the walls of the heart and the great vessels resulting from a rush of blood from a passage of relatively narrow calibre to one of much greater calibre causes a murmur. He categorically states that narrowing of a stream of blood without a fairly abrupt dilatation of calibre further on, does not cause murmur. Wood[5] accepts turbulence as the cause and mentions the possible causes of turbulence production as increased flow in hyperkinetic circulatory states, dilatation of ascending aorta or a stricture as found in co-arctation of the aorta.

If the theory of turbulence is accepted as a cause of murmur then it must be proved that turbulent energy in a fluid medium must always be converted into acoustical energy, so that the murmur becomes audible. Bruns[6] has put forth a very pertinent question based on careful experiments with fluids and obstructions to create artificial turbulences. According to him it is theoretically unlikely that a turbulence in blood will generate any significant acoustic energy. From the point of view of hydrodynamics the next possible source of sound in liquids is cavitation. This phenomenon fits very well with the audibility of stenotic murmur. Bruns[6] has experimentally shown that at least a driving pressure of 350 mm. of Hg is required to cause it.

After experimentally discarding turbulence and cavitation a new theory of causation of murmur has been put forward. When in a streamline flow of fluid an obstruction is placed, vortices are generated alternately from each side of the obstruction leading to formation of wake, which fluctuates periodically to at least R 200,000[6]. Depending on the type of obstruction a clear tone or a noise may only be produced. Careful experiments so far performed[6] all lead to the support of the periodic wake theory of causation of murmurs. This theory at the same time also gives plausible explanations of production of thrills, intensity and variations in the pitch of a murmur.

Clear explanations as to the cause of a musical murmur in the presence of rough irregular obstructions and in various congenital defects are wanted. Though this new theory sheds much light on various subjects of dispute regarding murmurs we shall have to keep an open mind and await further experimental confirmation from all possible angles.

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CURRENT MEDICAL LITERATURE

Psychoses and Systemic Lupus Erythematosus

O'CONNOR, J. F. (*Ann. Int. Med.*, **51** : 526, 1959) from the Department of Psychiatry, Columbia University College of Physicians and Surgeons, and the Presbyterian Hospital, New York, from a study of 40 patients with systemic lupus erythematosus observes :

Twenty-one had psychotic episodes during one or more hospitalisations, five had neurotic symptomatology, and 14 had no significant psychiatric symptoms. Fourteen patients had neurologic findings. The patients who became psychotic had a more severe form of systemic lupus erythematosus, as shown by the duration of their hospitalisations, severity of the medical symptoms, and the amount of steroid necessary to control these symptoms. Ten of the 11 cases that came to autopsy had evidence of brain damage. Of this group, all manifested either psychiatric or neurologic syndromes, or both. The occurrence of a psychosis during one hospitalisation did not contraindicate the reinstitution of steroid therapy. There was no evidence of alteration of medical and psychiatric syndromes; in fact, the reverse was true. The prognosis of the psychotic reactions was generally favourable. The development of a psychotic reaction is not an indication for the complete and immediate withdrawal of steroids, or for withholding steroids where medically indicated.

Pancreatitis after Chlorothiazide

JOHNSTON, D. H. AND CORNISH, A. L. (*J.A.M.A.*, **170** : 2054, 1959) from the Division of Medicine, Lexington Clinic, Ky., during the past one year report that acute pancreatitis was diagnosed in three women, all 50 years of age or older, who had been taking 0·5 to 1·0 g. of chlorothiazide daily for two or three months in the course of treatment for cardiovascular disease. In no case was there a history of conditions usually considered as predisposing to acute pancreatitis. The diagnosis was based on both clinical and laboratory studies and was confirmed in one case by fiistological study of autopsy material. Although the specific aetiology remained undetermined, chlorothiazide was considered as a possible cause. The authors wish to alert physicians to this possibility, which may warrant further investigation.

Haemorrhage from Multiple Sites in Chlorpromazine-Induced Jaundice

FLOEH, M. AND LEIBOWITZ, S. (*J.A.M.A.*, **170** : 2080, 1959) from the Medical Service, Beth Israel Hospital, New York, report that a 66-year-old diabetic woman bled from multiple sites (rectum, vagina, urinary tract, and skin) in association with jaundice induced by chlorpromazine, administered for 17 days. The likely explanation for the haemorrhage is hypoprothrombinaemia, although there is a possibility of defects in other coagulation factors. Note is taken that depressed prothrombin activity after chlorpromazine ingestion is uncommon. Haemorrhagic manifestations in association with the administration of chlorpromazine are rare. Particularly is this infrequent in reported cases of jaundice. Minor bleeding has been noted in the absence of jaundice, usually in the form of purpura. A striking clinical observation in this patient was the voracious appetite she manifested from the height of her illness.

Jaundice after Nicotinic Acid Therapy for Hypercholesteraemia

RIVIN, A. U. (*J. A. M. A.*, **170** : 2088, 1959) from the Medical Centre, University of California, Los Angeles, reports that a patient with familial hypercholesteraemia and tendon xanthomas exhibited marked reduction in serum chloresterol levels after therapy with large doses of nicotinic acid. The treatment was discontinued after 14 months because of the onset of jaundice, which probably represented intrahepatic cholestasis due to nicotinic acid toxicity.

Electroshock Therapy and Tranquillising Drugs

LESSE, S. (*J. A. M. A.*, **170** : 1791, 1959) from the Neurological Institute of the Presbyterian Hospital of New York and the Department of Neurology, Columbia University, gives in the following lines the summary of his observations :

Failures with intensive phenothiazine, reserpine, or meprobamate therapy do not, by any means, rule out the possibility of obtaining good results in psychotic patients for whom electroshock therapy (EST) is indicated. Of 170 psychotic patients who failed to benefit from the tranquillising drugs 69 per cent had satisfactory results with EST. Combined therapy with phenothiazines and electroshock, in general, neither reduced the total number of electroshock treatments required for good results, decreased the degree of pre EST apprehension, nor shortened the postconvulsive organic psychoses.

Parenterally given phenothiazines are helpful in managing some of the more agitated or manic patients in the intervals between electroshock treatments during the first few days of therapy. They also may be of aid in calming the agitation that may occur during an EST series and in the management of the agitation and restlessness associated with the post-EST organic psychoses.

Combined therapy, in the experience of the author, carries no greater risk than EST alone. The author does not find it necessary, in general, to discontinue the phenothiazine drugs before starting EST and does not recommend the routine use of electroshock therapy in combination with the phenothiazines, because it was not found to be more advantageous than EST alone, except in a few specially designated instances.

Small Doses of Vitamin K_1 for Correction of Reduced Prothrombin Activity

SCHIRGER, A., SPITTEL, J. A. AND RAGEN, P. A., (*Proc. Staff Meet. Mayo Clinic*, **34** : 453, 1959) from the Section of Medicine, Mayo Foundation write :

Small doses of orally administered vitamin K_1 effectively correct reduced prothrombin activity induced by coumarin drugs. The presence of severe liver disease makes the response to vitamin K_1 taken by mouth unpredictable. The dosage of vitamin K_1 must be indi-

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vidualised according to the level of prothrombin activity, the length of time the patient has received the coumarin drug, the desirability of continuing anticoagulant therapy, and the hazard of restoring the original risk of thrombosis.

Alimentary Bleeding of Obscure Origin

JONES, F. A., READ, A. E. AND STUBBS, J. L. (*Brit. M. J.*, **1**: 1138, 1959) from the Department of Gastroenterology of the Central Middlesex Hospital, London, from an analysis of a study of 1051 patients with haematemesis and melaena admitted between 1950-55, observe:

142 did not have any roentgenologic or other evidence of the source of bleeding. These 142 patients were followed up over a period of from 3 to 7 years. A positive diagnosis was subsequently made for 33 of them, but no diagnosis could be achieved for the remaining 109. Re-examination of the 33 patients with gastrointestinal bleeding of obscure origin revealed chronic gastric ulcer in 4, duodenal ulcer in 17, carcinoma of the stomach in 4, carcinoma of the colon in 3, carcinoma of the pancreas in 1, carcinoma of the small intestine in 2, Meckel's diverticulum in 1, and hereditary haemorrhagic telangiectasia in 1. Among the less common causes, special attention is drawn to pseudoxanthoma elasticum, von Willebrand's disease, diverticulosis of the colon, and the Mallory-Weiss syndrome. It was concluded that aspirin plays a definite part in precipitating gastroduodenal bleeding.

Patients with gastrointestinal bleeding of obscure origin should be investigated carefully. The personal history should be reviewed to find out if there had been any major emotional crisis in the few days preceding the bleeding. The patient's tendency to bleed and the possibility of recent medication with aspirin, cortisone or phenylbutazone should be considered as a cause; the mode of onset of the bleeding should be recapitulated, with special thought to the possibility of Meckel's diverticulum. Splenomegaly is constant accompaniment of portal hypertension and is masked only by obesity or ascites; in itself it raises the possibility of cirrhosis hepatis. The extent of further investigation will rest mainly on the clinical review of the case. Further barium meal, small intestine meal, and barium enema studies as well as liver function tests are necessary if findings of positive occult blood persist. A raised serum alkaline phosphatase value and dilated bile ducts may be found associated with an ampullary carcinoma. Laparotomy should be performed at the time of bleeding if positive findings persist. It is most important that the stomach should be opened and carefully inspected at the time of operation.

Bleeding Duodenal Ulcer

BANDLER, M. AND DESJARDINS, J. A. (*J. A. M. A.*, **170**: 2174, 1959) from the Division of Gastro-enterology, Department of Medicine, U.S. Naval Hospital, Philadelphia, from an analysis of a study of 162 bleeding duodenal ulcer patients with particular reference to pain, seasonal incidence and blood groups observe:

In a series of 162 consecutive cases of bleeding duodenal ulcer, the continued presence or exaggeration of pain compatible with duodenal ulcer disease was associated with minimal bleeding, whereas relief of pain was more characteristic of moderate and severe haemorrhage. Haematemesis by itself was not found to be a clear-cut indication of severe bleeding. Syncope, on the other hand, commonly heralded a massive haemorrhage. A history of upper gastrointestinal haemorrhage was of no apparent value in predicting the severity of bleeding from duodenal ulcer. The proportion of the number of severe haemorrhages to the total number of cases for a given decade increased progressively with advancing age. There appeared to be an increased frequency in the incidence of blood group O among patients with bleeding duodenal ulcer. An increased occurrence and severity of bleeding in the fall and winter months was also noted.

These findings suggest that greater stress should be placed on the pain pattern in patients with bleeding duodenal ulcers. The "silent bleeder" or the one in whom ulcer pain has been relieved with bleeding would be more likely to have a more severe haemorrhage and therefore, would demand a more vigorous therapeutic approach.

Delayed Haemorrhage After Kidney Biopsy

FELTON, L. M. AND ANDRONACE, J. M. (*J. A. M. A.*, **170**: 2185, 1959) from the Department of Urology, New York Medical College, Metropolitan Medical Centre write that a case of delayed bleeding occurring after a percutaneous biopsy of a kidney was unusual in that the patient did well and apparently had no ill-effects from the biopsy until the ninth day after the procedure, when he suddenly went into profound shock secondary to a delayed rupture of the kidney from which the biopsy specimen was taken. The resultant haemorrhage jeopardised the patient's life and necessitated an emergency nephrectomy. The renal biopsy is a recognised diagnostic procedure. However, the patient should be carefully observed for two weeks thereafter. As more biopsies are attempted, more such cases are likely to occur. An awareness of the possibility of delayed bleeding will facilitate its early diagnosis and treatment.

Eosinophilic Peritonitis

HARLEY, J. B., GLUSHIEN, A. S. AND FISHER, E. R. (*Ann. Int. Med.*, **50**: 301, 1959) from the Department of Medicine and Physiology, Veterans Administration Hospital, Pittsburgh, and the University of Pittsburgh School of Medicine in reporting on the clinical and pathologic findings in four patients with eosinophilic peritonitis observe:

The varied clinical manifestations of this disorder are emphasised. Operative intervention or use of adrenal steroids failed to alter the clinical course significantly. Although there is suggestive evidence that eosinophilic peritonitis may be allergic in nature, absolute proof for such aetiology is lacking. Diagnosis, in the instances where ascites is present, may be facilitated by the observation of a large number of eosinophils in the ascitic fluid.

CURRENT TOPIC

GOOD-WILL VISIT TO THE I.M.A. BY REPRESENTATIVES OF THE B.M.A.

Dr. S. Wand, Chairman of Council and Dr. D. P. Stevenson, Secretary of the British Medical Association, paid a good-will visit to the I.M.A. and were in India from November 8 to 20, 1959. They visited branches at Delhi, Jaipur, Calcutta, Bombay, Hyderabad, Bangalore and Madras. Unfortunately, they were unable to spend more time in India due to unavoidable reasons, though they had intended originally for a longer visit. In the short period of time they had had at their disposal, it was not possible for them to visit more branches, which they regretted very much.

Dr. S. Wand said at most of the places that all over the world, Governments had intruded into the field of medical care and with this, politics had entered the profession.

Some decades ago, he said, medicine was practised by individuals more or less in isolation. Vast changes had now taken place. With increasing specialisation had arisen the need for teamwork. The cost was also increasing. At the same time Governments realised that medical care of the people could not be left entirely to individuals.

Many "old-fashioned" doctors criticised the intrusion of politics in the profession. But in the present circumstances it was difficult anywhere in the world to keep medicine out of politics. Governments and local organisations, which allotted money, wanted to know how it was spent. At the same time, the profession had to be protected from the wiles of those who did not understand the practice of medicine.

Dr. Wand said that in Britain the intrusion of the State in the field of medical care began in 1911, with the passing of the National Insurance Act. A big step forward was the nationalisation of health services in 1948 which entitled every citizen to obtain medical care without any direct payment. The cost of the services was about £600 million a year.

Dr. Wand and Dr. Stevenson further impressed on the members of the Association that for obvious reasons, the B.M.A. did not approve of the question of appointment of full-time medical teachers in clinical subjects. After the National Health Scheme was put into operation, the Government of U.K. consulted the B.M.A. and appointed just a few medical personnel on full-time and non-practising basis particularly for promotion of research work in teaching medical institutions. The B.M.A. was definitely of opinion that it would not be in the best interests of the patients as well as medical students, if all the teaching appointments in various clinical subjects were attempted to be made whole-time and non-practising. In India, they considered such a scheme could not be accepted, as the number of the hospital beds was grossly inadequate and the Government could not afford to pay adequate salaries to the medical personnel if they were appointed on a full-time basis.

Dr. Wand and Dr. Stevenson also thought that it would be wiser for the Government to consult the I.M.A. from time to time to discuss these and other relevant matters.

Referring to the rights won by doctors in Britain, he spoke of the close contacts established between the Government and the B.M.A. There had hardly been an occasion when the Government declined to accept the advice given by the B.M.A. so far as health services were concerned. He expected that similar relations should be established in India between the Government and the I.M.A.

Dr. D. P. Stevenson, Secretary of the B.M.A., spoke on how his organisation strove to maintain the improved medical standard and ensure that doctors' rights were not interfered with. The B.M.A. was founded 127 years ago and it had seen the development of the practice of medicine in Britain from an entirely unorganised state to the efficient system of today.

• • •

Given below is a summary of their programme at various places :

Delhi—November 8, 1959 :

Due to bad weather at Moscow, they were unable to reach Delhi on November 7, as scheduled and instead arrived nearly 30 hours late in the late afternoon of November 8. Consequently, they were unable to attend the reception meeting arranged for them by the Delhi branch or the private dinner arranged by the Hony. General Secretary at the Delhi Gymkhana Club. They were, however, able to attend a private cocktail party arranged by Dr. Ved Prakash, the Hony. Joint Secretary at his house on November 8, where they were able to meet nearly 100 members of the medical profession in Delhi and their wives. At this function, they were able to meet the office-bearers of the Central Office and the Delhi branch also.

THE GUESTS AT A COCKTAIL PARTY ARRANGED BY DR. VED PRAKASH, HONY. JOINT SECRETARY, I.M.A., AT DELHI (*L—R*) : Dr. Ved Prakash, Mrs. A. P. Mittra, Mrs. Ved Prakash, Dr. S. Wand, Dr. A. P. Mittra (Hony. General Secretary, I.M.A.) and Dr. D. P. Stevenson

A meeting for the visitors with the Union Health Minister could not be made possible because of his being out on tour.

Jaipur—November 9, 1959 :

A day's visit to Jaipur was arranged through the courtesy of the Jaipur Branch where a reception meeting was held which was addressed by the visitors. They were also entertained to lunch by the Branch and some sight-seeing was arranged for them including a visit to the Medical College Hospital and the Jaipur Museum.

Calcutta—November 10-12, 1959 :

Dr. S. Wand and Dr. D P. Stevenson reached Calcutta by air at Dum Dum at 10-5 A.M. They were received by the President, Bengal Provincial Branch; Vice-President, Calcutta Branch; Editor, Journal of the I.M.A.; Jt. Secretary, I.M.A. (Central) at Calcutta; Secretary, Calcutta Branch; Secretary and Jt. Secretary, Bengal Provincial Branch and some members of the Executive Committee of the Calcutta Branch.

From the Air Port they were conducted to Great Eastern Hotel where the Association arranged for two air conditioned rooms. On the same day at 1-0 P.M. Dr. Wand and Dr. Stevenson were entertained to lunch at the Calcutta Club which was subscribed by some members of the Calcutta Branch. At 2-30 P.M. they were taken round the city as a part of the programme for sightseeing. The Hony. Secretary, Calcutta Branch, accompanied them. At 4-30 P.M. they arrived at the I.M.A. Office and were entertained to tea at which several other members of the I.M.A. joined. At 7-0 P.M. a meeting of the medical profession was arranged which Dr. Wand and Dr. Stevenson addressed. They dealt on the organisational points of the B.M.A. and answered questions put by some of the members of the audience.

At night they attended a dinner given by the Editor and the Commitee of the Journal of the I.M.A. at the Chinese Pavillion at the Great Eastern Hotel. Dr. P. K. Guha, Editor of the Journal and Dr. N. C. Banerjee, Secretary, were all attention to the guests.

THE GUESTS ON ARRIVAL AT THE DUM DUM AERODROME

(*L—R*)—Major K. K. Ghosh, Dr. D. Mukherjee, Dr. M. Nandi, Dr. B. Ukil, Dr. D. N. Roychoudhury, Dr. D. P. Stevenson, Lt. General D. N. Chakrabarti, Dr. S. Wand, Dr. T. Banerjee, Dr. B. P. Tribedi, Dr. P. K. Guha, Dr. B. Roy and Dr. S. Dutt.

DR. B. P. TRIBEDI, PRESIDENT, I.M.A., BENGAL STATE BRANCH AND DR. H. K. ROY, PRESIDENT, I.M.A., CALCUTTA BRANCH, ENTERTAINED THE GUESTS AT THE RECEPTION DINNER PARTY AT THE GREAT EASTERN HOTEL, CALCUTTA

Facing camera (*L—R*) : Gen. D. N. Chakrabarti, Director of Health Services, West Bengal, Dr. H. K. Roy, Dr. D. P. Stevenson, Dr. B. P. Tribedi (*standing*), Dr. S. Wand, Major K. K. Ghosh, Vice-President, I.M.A.—Bengal, Lt. Col. N. C. Chatterji, Jt. Director of Health Services, West Bengal, Dr. P. K. Guha, Editor, *Journal of the I.M.A.* and Dr. J. C. Banerjea. Among others are : Dr. R. C. Sen, Vice-President, I.M.A.—Bengal, Dr. N. B. Roy, Dr. Souren Sengupta, Hony. Provincial Secretary, I.M.A.—Bengal, Major General A. K. Gupta and Dr. J. B. Chatterjea

DR. S. WAND ADDRESSING THE MEMBERS AT THE I.M.A. HALL, CALCUTTA

November 11—In the morning they visited S. S. K. M. Hospital where they had a discussion on the different aspects of hospital managements with Major General A. K. Gupta, the Superintendent of the Hospital.

They met the Chief Minister of West Bengal, Dr. B. C. Roy, at the Writers' Buildings. They had discussions covering a wide range of subjects particularly in regard to the role of medical association *vis-à-vis* Goverament.

Dr. Wand and Dr. Stevenson were then taken to the Institute of Child Health where they were received by the Director of the Institute, Dr. K. C. Chaudhuri. They went round the various departments of the Institute and expressed their appreciation with regard to the work done there. Their next visit was the School of Tropical Medicine where they were received by Dr. R. N. Chaudhuri, Director. They were accompanied by Dr. N. C. Banerjee, Dr. M. Nandi and Dr. S. Dutt in course of their visit round the city hospitals.

On the same day they were entertained to a lunch at the Calcutta Club given by Dr. Sengupta, Secretary, *Indian Medical Forum*, a monthly medical journal published from the city. Various cross-sections of the public including eminent medical men were present at the lunch.

In the afternoon Dr. Wand and Dr. Stevenson were entertained at a tea party given by Dr. A. B. Roy, Minister of Health, Government of West Bengal. They went to visit Woodlands Nursing Home accompanied by Dr. Davis. At night they were entertained at a dinner party given by Dr. B. P. Tribedi and Dr. H. K. Roy, Presidents, Bengal Provincial Branch and Calcutta Branch respectively, at the Great Eastern Hotel. Many notable dignitaries were present at the party.

November 12—They were taken round to some places of the city and reached Dum Dum Air Port from where they emplaned. They were given a hearty send off by the members of the Indian Medical Association. Among those present were : Dr. P. K. Guha, Editor, *Journal of the I.M.A.*, Dr. N. C. Banerjee, Hony. Secretary, *Journal of the I.M.A.*, Dr. M. Nandi, Hony. Secretary, I.M.A., Calcutta Branch, Dr. D. Mukherjee and Dr. T. Banerjee, members of the Executive Committee, Calcutta Branch.

During their stay at Calcutta, Dr. Wand and Dr. Stevenson were guests of the Indian Medical Association, Bengal Provincial Branch and Calcutta Branch.

Bombay—November 12-14, 1959 :

Dr. Wand and Dr. Stevenson were received at the Air Port by Dr. C. S. Thakar and Dr. A. M. Shah, Dr. A. C. Mehta and Dr. M. A. Panwala, at 4-30 P.M. on 12 November 1959. The guests were taken to see the Aarey Milk Colony of Government of Bombay and Vihar Lake and its gardens by Dr. A. C. Mehta and Dr. M. A. Panwala. They were greatly impressed by the Aarey Milk Colony in its endeavour to supply pure milk to the public.

On the 13th November, Dr. S. C. Sheth met them at the Taj Mahal Hotel at 9 A.M. and took them round the B. Y. L. Nair Hospital and from there to J. J. Group of Hospitals. At J. J. Group of Hospitals, Dr. R. V. Sathe and Dr. S. C. Seth took them round the various departments.

From there they were taken to the K. E. M. Hospital where Dr. M. J. Shah, President, Bombay Territorial Branch, received them and introduced them to the Dean, Dr. S. V. Joglekar, and showed them the working of the various departments of the hospital. From there they were taken to Haffkine Institute where they were received by Dr. H. I. Jhala, Director, who showed them various research departments and the snake farm. The guests were much impressed by the advancement in medical sciences at the various institutions.

They were entertained to lunch by Dr. M. J. Shah at K. E. M. Hospital.

At 3 P.M. at G. S. Medical College, they addressed the members of the medical profession on "Some Aspects of Medical Profession in U.K. and National Health Services in U.K."

The meeting was presided over by Dr. R. V. Sathe and was attended by a large number of practitioners and the guests gave a very informative talk on the subject. At the end of the talk they answered a number of questions on B.M.A. organisation and also on the National Health Services in U.K. The vote of

MEMBERS WHO PARTICIPATED IN THE DINNER PARTY AT THE WILLINGDON SPORTS CLUB, BOMBAY, ON 13-1-59
Fifth from right is Dr. D. P. Stevenson and next to him are Dr. M. J. Shah, Dr. S. Wand and Dr. N. N. Kailas, Dy. Minister of Health, Government of Bombay

DR. S. WAND REPLYING AT THE DINNER AT THE WILLINGDON SPORTS CLUB, BOMBAY

DR. D. P. STEVENSON REPLYING AT THE DINNER AT THE WILLINGDON SPORTS CLUB, BOMBAY

thanks was proposed by Dr. S. C. Sheth. The meeting was followed by tea when the guests talked and discussed with the individual members of the profession in a personal and friendly way, on the working of Health Insurance Scheme in Bombay and U.K.

From there the guests were taken to the town for sightseeing and marketing.

At 8-30 P.M. at Willingdon Sports Club they met the Dy. Minister for Health Dr. N. N. Kailas and discussed with him various medical problems. At 9 P.M. they attended the dinner at the same place by the Bombay Territorial Council. Dr. M. J. Shah introduced the guests to the members before the dinner.

At the dinner Dr. M. Shah stated that the I.M.A. was thankful to the B.M.A. for its co-operation and help in all affairs in the working of the Association and B.M.A. being the premier organisation, the I.M.A. had to learn from it a lot. The guests in their reply thanked

the members of the I.M.A. for their hospitality and stated that they were much impressed by the various advances made by the medical profession and the medical sciences in Bombay and desired that any one visiting U.K. would make it a point to visit the B.M.A. House in London and meet the Secretary, B.M.A. personally and assured that B.M.A. would do its best for any member of the I.M.A. visiting London.

On 14th November 1959, Dr. C. O. Karunakaran, President, I.M.A., made a special effort to come to Bombay and met the guests at the Air Port at 7 A.M. and Dr. A. M. Shah of Bombay arranged the meeting between Dr. Karunakaran and the guests at the breakfast table at the Air Port as the guests had to leave Bombay that morning.

Hyderabad—November 14-16, 1959:

The Andhra State Branch, and the Hyderabad and Secunderabad Branches jointly arranged for the reception of the visitors, and the programme at Hyderabad included a reception meeting in the I.M.A. Building, Hyderabad, a banquet and other entertainments. They were entertained to lunch by the members of the Executive Committee, Andhra State Branch, I.M.A. A dinner was also arranged at the Secunderabad Club. They were able to visit the Niloufer Hospital and the Radium Institute, the Osmania Hospital and the Osmania University, and were officially received at the K. E. M. Hospital, Secunderabad. Suitable arrangements were made by the branches for the guests for cultural entertainment and sight-seeing.

Bangalore—November 16-18, 1959:

From Hyderabad Drs. Wand and Stevenson reached Bangalore on the evening of November 16. Here also, there was a largely attended reception meeting held by the Bangalore Branch in their honour. At another function the guests addressed the students of the Bangalore Medical College. Visits were arranged for them to the several hospitals in Bangalore, the Indian Institute of Mental Health, the S. D. S. Sanatorium and the Indian Institute of Sciences. They were received by Dr. K. K. Hegde, Minister of Health, Mysore State at the Vidhana Soudha. Lunches and dinners were arranged in their honour by the I.M.A. in Bangalore.

Madras—November 18-20, 1959:

It was on Wednesday (18-11-59) evening that a representative gathering of the Madras City Branch of the Indian Medical Association awaited at the Aerodrome the arrival of Drs. S. Wand, Chairman of the Council and D. P. Stevenson, Secretary of the British Medical Association.

This was preceded by feverish preparations for housing the guests and formulating a colourful programme for them. The necessity for such an anxiety was evident. They were representing a sister organisation from a land which had contributed much to medicine and medical men of this country. One of the distinguished members of this Branch, the Speaker of the Legislative Assembly, Dr. U. Krishna Rau had also come to the aerodrome and special permission was granted to the members to enter the enclosure to receive the distinguished guests. They were accompanied by the President of the State Branch, Dr. T. V. Sivanandam.

The guests were immediately driven to the Hotel Connemara where accommodation for them was arranged. From the hotel they were taken round the city's most sought after evening resort—the Marina. They appreciated the Marina very much. They were then brought to Hotel Dasaprakash where a cultural show awaited them. This show consisted of dances by Baby Sasikala, daughter of the Treasurer of the branch, Dr. K. V. Swamy. This little girl who was hardly nine years old captured every one's heart and the visitors were no exception. Dr. S. Wand gave his impression rather humorously. He said that the little dancer would create a stir in the minds of any New-Yorker or Londoner. He volunteered to act as an agent on a small commission only if Dr. Swamy could send his daughter to U.K. or America! Kumari Vimala, daughter of the President, Dr. G. Sriramulu, filled up the intervals rendering in her melodious voice, songs in almost all the major languages in vogue in India.

The dinner was very well liked by the guests. It was purely vegetarian as a good majority of the guests wanted it to be, so that the visitors could taste some South Indian dishes.

After dinner Dr. K. C. Nambiar, one of the senior and respected members of the branch, stated that all degrees obtained in India should be recognised by the U.K. Medical Council. The visitors assured that though they were not connected with the British Medical Council they would convey this request and do what they could.

Thus ended the first day.

It was programmed for the guests to visit the city hospitals the following morning (19-11-59). But only one hospital—The Cancer Institute—situated at picturesque Adyar could be visited. Dr. Nathamuni Naidu, Vice-President of the State Branch, also accompanied the party.

THE GUESTS ON A VISIT TO THE CANCER INSTITUTE, MADRAS

(*L—R*) *Front row*: Dr. V. Shanta; Dr. G. Sriramulu, President, Madras City Branch; Dr. S. Wand. *2nd row*: Dr. C. Nathamuni Naidu, Vice-President, Madras State Branch, Dr. D. P. Stevenson, Dr. A. Pattabhi, Dr. D. R. Varman, Secretary, Madras City Branch. *Back row*: Dr. S. Krishnamurthi, Director of the Institute.

The visitors were so engrossed in the work done there, by and under the able guidance of Dr. S. Krishnamurthy, the Institute's Director, that time fleeted without anybody noticing it. The party could get out only at 11 and so visits to other hospitals had to be dropped as there was an engagement with the Health Minister, Shri M A. Manikavelu at Fort St. George at 11-30 A.M.

The guests discussed with the Minister matters pertaining to Health for about 30 minutes. Following this, they had tea with Dr. U. Krishna Rau at his chamber in Fort St. George. The visitors were then taken to Mahabalipuram by the State Joint Secretary Dr. B. Rama Rao. They liked the place very much and spent quite a good time there, so much so, they were late for the evening programme of tea and meeting.

A sizable audience had collected to hear the visitors from U. K. Dr. G. Sriramulu, President, introduced the visitors to the gathering. Dr. Wand spoke about various topics including the National Health Services. Dr. Stevenson gave a vivid account of the birth and growth of the B.M.A. and said it was a powerful body having about 97 per cent of doctors in its fold. He also said that the Government always took their advice in matters pertaining to health, medical relief and medical education.

On 20-11-59 the office-bearers of the City Branch saw the visitors off at the aerodrome. They were leaving for Ceylon.

NOTES AND NEWS

General Assembly of W.M.A.

The German Medical Association, host of the XIVth General Assembly of The World Medical Association, scheduled to convene in West Berlin, September 15-22, 1960, has extended a cordial invitation to all the doctors of the world to attend this outstanding meeting.

The Bundesaratekammer (German Medical Association) will convene its 1960 Annual Meeting concurrently with the convening of the General Assembly. This is the first time that a host medical association to The World Medical Association has scheduled its own Annual Meeting in conjunction with the meeting of The World Medical Association. The two associations will meet jointly in their opening and closing plenary sessions. All activities including the Postgraduate Teaching Film Programme, exhibits and scientific sessions will be held in one building in order to enable doctors to attend the various sessions associated with each meeting which are of special interest to them.

Additional information including programmes and schedules will be available on or about March 1st at the W.M.A. Headquarters Secretariat. Request should be addressed to: The World Medical Association, 10 Columbus Circle, New York 19, New York.

Asiatic Congress of Obstetrics and Gynaecology

The Second Asiatic Congress of Obstetrics and Gynaecology will be held in Calcutta on 23-25 January, 1962. The subjects for discussion are (1) Malignant Tumours of the Female Genitalia, (2) Toxaemia of Pregnancy, (3) Maternal and Perinatal Mortality.

Detailed information from the Hony. Secretaries, Steering Committee, Second Asiatic Congress of Obstetrics and Gynaecology, 4, Chowringhee Terrace, Calcutta 20, India.

Live Poliovirus Mass Vaccination in U.S.S.R.

The Soviet Union has embarked on a mass vaccination programme against poliomyelitis using a live-virus vaccine given by mouth. Over 10 million persons have already been vaccinated; fifteen million were to be vaccinated by January 1960, and it is planned that, by the end of 1960, 88 million persons will have been vaccinated including the entire population of the Soviet Union under the age of 20.

Live-virus vaccine against polio was developed by Koprowski, Sabin, Cox and others in the United States, and differs from the widely used Salk vaccine, which is based on killed virus and is given by injection. The vaccine used in the U.S.S.R. is made from virus strains supplied by Dr. Sabin of Cincinnati.

In agreement with the Soviet Ministry of Health and the U.S. Public Health Service, the World Health Organisation invited the distinguished American scientist, Dr. Dorothy M. Horstmann, to visit the Soviet Union in order to evaluate the safety and efficiency of the new vaccination against poliomyelitis. Dr. Horstmann, who is Associate Professor of Preventive Medicine and Pediatrics at the Yale Scilbol of Medicine, stayed in Moscow, Leningrad, Tashkent and Riga and then visited Poland and Czechoslovakia, where live polio-virus vaccine is also being used.

According to Dr. Hortsmann's report, the marked reduction of poliomyelitis in the Soviet republics where oral vaccination has been applied suggests that the vaccine has played a significant role in reducing the incidence of paralytic poliomyelitis. Compared to the pattern of rising incidence over the past few years, the low 1959 incidence in the U.S.S.R. is impressive. Dr. Horstmann says, however, that at least another year is needed before a valid judgment can be made about the effectiveness of the vaccine.

Principles of Social Security and Medical Care

The twelve principles of Social Security and Medical Care adopted by the Second General Assembly of The World Medical Association, Geneva, Switzerland, 1948 and reaffirmed by the Fourth General Assembly in 1950, the Sixth General Assembly in 1952 and the Ninth General Assembly in 1955, read as follows:

Whenever medical care is provided as part of social security, the following principles should govern its provisions:

I. Freedom of choice of physician by the patient, Liberty of physician to choose patient except in cases of urgency or humanitarianism.

II. No intervention of third party between physician and patient.

III. Where medical service is to be submitted to control, this control should be exercised by physicians.

IV. Freedom of choice of hospital by patient.

V. Freedom of the physician to choose the location and type of his practice.

VI. No restriction of medication or mode of treatment by physician except in case of abuse.

VII. Appropriate representation of medical profession in every official body dealing with medical care.

VIII. It is not in the public interest that physicians should be full-time salaried servants of the government or social security bodies.

IX. Remuneration of medical services ought not to depend directly on the financial condition of the insurance organisation.

X. Any social security or insurance plan must be open to the participation of any licensed physician, and no physician should be compelled to participate if he does not wish to do so.

XI. Compulsory health insurance plans should cover only those persons who are unable to make their own arrangements for medical care.

XII. There shall be no exploitation of the physician, the physician's services or the public by any person or organisation.

Republic Day Honours for Medical Men

Dr. R. N. Chaudhuri, Director, School of Tropical Medicine, Calcutta, and Dr. V. N. Shirodkar, Gynaecologist, Bombay, have been awarded "Padma Bhusan" on the Republic Day (January 26), 1960. Dr. K. R. I. Doraiswami, Director, the Premier Radiological Institute and Cancer Hospital, Mylapore, Madras, has been awarded "Padma Shri".

It may be recalled that two eminent medical men of Bombay, Dr. R. J. Vakil and Dr. J. C. Paymaster, were awarded "Padma Bhusan" last year.

Foreign Distinctions for Medical Men in India

At a quarterly meeting held on November 3, the following Indian doctors were elected Fellows of the Royal College of Physicians of Edinburgh: Dr. B. Banerjee, Dr. K. S. Mathur, Dr. R. Natarajan, Dr. T. J. Danaraj, Dr. D. P. Basu.

Dr. M. G. Varadarajan has been elected a Fellow of the College of Chest Physicians (USA).

Synthetic Drug Factory

Details regarding the Russian-aided Rs. 8·5 crores Synthetic Drug Factory are being worked out.

A nine-man Russian team headed by Mr. Valachek arrived at Hyderabad to inspect the site and examine the possibility for the disposal of the factory effluent. Mr. N. Srinivasan, Industrial Adviser (Chemicals) and Dr. R. Shaw, Development Officer (Drugs), Government of India are with the team.

The Synthetic Drug Factory will have a capacity of producing 900 tons of drugs per year and will employ 1,700 hands. The quantum of Russian loan assistance in the factory is Rs. 2·3 crores. A final decision about the location of the factory will be taken on the basis of the Project Report of the Russian team.

The Russian team inspected the sewage system in Hyderabad with a view to assessing how far the effluent from the factory could be disposed of without affecting the crops and health of the population. Certain alternatives were discussed with the State officials. Those who were present at the discussion with the Russian team included the Director of Industries, Andhra Pradesh Government, the Superintending Engineer, P.W.D. (Headquarters Circle), the Superintending Engineer, Local Administration and Public Health and the City Corporation officials connected with drainage.

Sharp Drop in Cholera Deaths in India

India had a record low number of cases of cholera in 1959, according to the World Health Organisation.

In its weekly epidemiological record published at Geneva on 22-1-60, the Organisation says the cases notified were very much less as compared with 67,000 in 1958.

About 60 per cent of all cases occurred in the Lower Ganges Valley but the incidence was very low in West Bengal—4,055 last year against 12,334 in 1958—and there were less than 200 cases in Orissa—192 compared with 5,393.

In Delhi, Himachal Pradesh, Kerala and the Punjab there were no cases notified and one of the greatest drops in cases occurred in Andhra Pradesh with a 1959 total of 1,224 against 10,758 for the preceding year.

Additional U. S. Grant to India for Malaria

Additional assistance of Rs. 30·6 lakhs (dollars 665,580) has been authorised by the United States for India's malaria eradication programme.

A supplemental agreement signed on 22-1-60 between the Government of India and the U. S. Technical Co-operation Mission (T.C.M.) will provide India special funds to procure chloroquine and primaquine phosphate tablets, 150 jeeps, microscopes, slides, and calculating machines. These will be purchased outside India and will further aid the eight-year old Indo-U.S. efforts to banish malaria from India and afford full protection to the entire nation.

The latest agreement brings the total of US dollar assistance to the malaria control and eradication programme to more than Rs. 29 crores (dollars 6·5 million). This is in addition to the PL 480 grant of Rs. 8 crores made last year.

The present agreement was signed by Shri N. C. Sen Gupta, Joint Secretary, Union Ministry of Finance and Mr. C. Tyler Wood, T.C.M. Director.

New Substitute for Blood in Emergency

Two Japanese surgeons reported on 11-1-60 that water containing a chemical obtained from the algae in giant brown seaweed could be used as a substitute for blood in emergency transfusions.

Dr. Masanobu Tomoda and Dr. Kyoshi Inouchi in the *Journal of the International College of Surgeons* said

the solution had proved in their experiments to be superior to ordinary salt water or sugared water in preventing shock during operations or after severe burns as it did not break down in the bloodstream.

Efforts were being made to find a plentiful substance which can be substituted for whole blood or blood plasma in treating the survivors of a major disaster, such as an atomic attack. They reported that they had used the chemical successfully in 192 abdominal operations.

National Tuberculosis Survey

The national tuberculosis survey has revealed that about 5 million people in India, or 1·3 per cent of the population, suffer from pulmonary tuberculosis. The total number of cases is thus much larger than was presumed. The number of infectious cases will be at least 1·5 million, that is 0·4 per cent of the population.

The fact that there is such a large number of infectious cases in urban and rural areas, the survey report says, is a matter of serious concern and demands the highest priority for the anti-tuberculosis campaign in the country's five-year plans.

The survey has also belied the earlier assumption that the population in urban areas was more affected by TB than in rural areas. For this reason, programmes for control of tuberculosis have to be extended to rural areas also in the Third and subsequent Plans.

Since the sample survey covered only 40 per cent of the total population spread over six zones, the estimates cannot be strictly used for the country as a whole, says the report.

REVIEWS

Modi's Medical Jurisprudence and Toxicology—Edited by N. J. Modi. 13th ed., 1959. N. M. Tripathi Private Ltd., Bombay 2. 6¼" × 9½", pp. 808, price Rs. 25/-.

Modi's Medical Jurisprudence is undoubtedly the standard text-book on Forensic Medicine in India.

This edition is a distinct improvement over the previous one, as it shows, much re-writing and addition of new materials. New cases of medicolegal importance have been incorporated.

The book contains everything that is required to be known to the medical students and practitioners and members of the legal profession.

Being written with the background of Indian conditions in view, the book has acquired a special importance in our country.

Surgery of the Foot—By Henri L. DuVries, M.D. With a Foreword by Karl A. Meyer, M.D. and an Introduction by Edward L. Compere, M.D. 1959, The C. V. Mosby Company, St. Louis. 9¼" × 6½", pp. 494, price $12.50.

To quote Wood Jones, "Man's foot is all his own. It is unlike any other foot. It is the most distinctly human part of the whole of his anatomical make-up." But there is a distinct void in the ordinary text-book teaching of the surgery of the foot and also in the number of reference books on the subject, as compared to that on the surgery of the hand. So this book containing 403 illustrative figures and the lucid exposition of common but difficult problems by one who has made the foot his life's study is most welcome.

The chapters among others include structure and function, examination and diagnosis, the foot in diabetes, disease and deformities of toenails, diseases of nerves, static deformities, anomalies and congenital defects, operative principles, infection of the foot, fractures and dislocations and amputations. These will be useful both to general and orthopaedic surgeons. Specialists will appreciate chapters or disorders of tendons and sesamoids, aseptic necrosis, diseases of synovia and fascia, tumours, cysts and exostoses. Chapters on topical subjects as burns, sprains and athletic injuries are useful and instructive. In fact there is hardly any aspect of the foot which has not been covered. The author should be complimented for providing such a good reference book on surgery of the foot.

Essentials of Biochemistry—By S. M. Banerji, M.SC., M.B. 1959. S. Bhattacharya & Co., Calcutta 13. 8½" × 5½", pp. 174, price Rs. 7·00.

The volume is Section II of *Essentials of Human Physiology* by the author. It deals with biochemistry and biophysics, catering to the needs of preclinical medical students. The book is unfortunately sketchy and lacks in accuracy and precision, and is open to criticism, for it does not point to "the proper appreciation of principles governing physiological phenomena" (Preface).

The author has defined organic chemistry as chemistry of *combustible* carbon compounds (p. 1). Carbon compounds are not always combustible, e.g., carbon dioxide, carbon tetrachloride; still they are enlisted in organic chemistry. He gives an incomplete classification of the types of organic compounds (p. 2). He does not mention amines (needed for the study of proteins), esters (for fats), unsaturated compounds (for fats, essential acids, vitamins), although he states that a knowledge of the chemistry of such type compounds is essential for understanding chemistry of proteins, carbohydrates, etc. Alcohols have been classified as 'mono-atomic' (p. 3), which unfortunately is inaccurate. The general formula for primary alcohols should have been $C_nH_{2n+1}CH_2OH$.

"Like *alkaline hydrates*, alcohols react with acids" (p. 4), is not a precise statement. The definition of fat as an ester of cholesterol with higher fatty acids (p. 6) needs revision. Carbohydrates have been stated "as aldehydes and ketones of polyhydric alcohols", which is a faulty expression. Proofs of the cyclic structure of glucose are not at all precise (p. 9). The most important proofs have been omitted.

d-Arginine, which is *alpha*-amino-*delta*-guanidinovaleric acid, has been noted as ornithine+urea (p. 21). The structural formula for proline (p. 23) has been wrongly drawn.

Unfortunately such expressions as the following are not accurate :

"For one and the same substance (should be solution) at constant temperature, osmotic pressure varies as concentration", "One gm.-mol of a gas occupies a litre at 22·4 atmospheric pressure" (add at 0°C) (p. 27); "cH is one or 10^0 (mention unit, gm. ion) in one litre" (p. 32).

"Buffer salts" (p. 34) (should be solution).

". . . electric current will flow from greater (H+) to lesser (H+) and can be measured by a potentiometer" (p. 35). (Surely a potentiometer will not measure a *current*).

These are but a few instances of very many examples of inaccuracies in the exposition of the most elementary fundamental principles of chemistry on the basis of which the chemical reactions in body processes can be appreciated. The metabolism of the food principles has been mentioned in bare outline.

The book may serve as a cram-book during examination, for last-minute brushing up of the more important points of topical subjects on biochemistry for preclinical medical students.

Punjab Medical Journal, Vol. IX, Nos. 1 and 2, Special Current Therapy Numbers, Jullundur City, price Rs. 2/- (exclusive of postage) for each.

There are altogether 32 articles. The very first article in part I, devotes more than ten pages on diagnosis. The therapy discussed includes both old abandoned remedies and newer ones. It is curious that in a therapy number no special stress has been given to the dosage schedule.

A smaller dose of salicylate has been recommended in the treatment of acute rheumatic attack, because the author is of opinion that our patients tolerate comparatively smaller doses. The reviewer does not agree. Salicylate in the dose of 120 grains a day is quite well tolerated and a dosage less than that will not maintain a proper effective blood level, when combined with sodium bicarbonate.

In the article on treatment of cardiac arrhythmias the authors have started with the classifications of tachycardias, bradycardias and irregular rhythms, but have switched on to discuss therapy on the pharmacological basis. Here again dosage schedule has been omitted in the case of some of the drugs mentioned.

The reviewer fails to follow the rationality of inclusion of articles on traumatic lesions of the hand, effects of heat, histamine, haemangioma, bulbar palsy, vaccination against smallpox, and psychological therapy in Current Therapy numbers. The choice of the heading "Current Trends in T.A.B. Therapy" to discuss therapy in enteric fevers is not happy, as T.A.B. reminds one of the vaccine and not the diseases.

On the whole the majority of the articles give current trends in management and therapy. There are only a few brilliant articles.

Shree M. P. Shah Medical College Magazine, Vol. I, No. 1, July 1959. Editor—Shri Sobhag M. Shah. pp. 70+iii.

The students of the M. P. S. M. College are congratulated for being able to bring out a college magazine. The reviewer regrets his inability to find out whether the magazine will be an annual feature or other issues may come out at periodical intervals.

The practice of publication of messages with or without photographs (perhaps the Editor was not obliged with one in spite of his best efforts) is universal. But what about the messages? Sri C. S. Patel's message does not contain a single reference to the magazine, while Sri Y. B. Chavan saw the publication of the College Magazine on 22-4-59 though the magazine saw daylight three months later and Sri U. N. Dhebar writes a mere personal history.

The contributions by the students are quite good. A few minor lapses may be pointed out so that in future better care may be bestowed before publication. Are cholinesterases neurohormones? 'Memorium' ought to have been spelt as Memoriam and 'berieved' as bereaved.

Biochemistry of Antibiotics—Edited by K. H. Spitzy and R. Brunner. (Volume V of the Proceedings of the IVth International Congress of Biochemistry, Vienna, 1958). Pergamon Press, London. 6" x 9", pp. 230, price 68s. net.

The book, the fifth volume of the series of 18 volumes, published by the International Union of Biochemistry, is a collection of papers read by specialists on different aspects of biochemistry in the symposium V of the Congress. Altogether 18 papers with discussion, have been reported, of these some are of special interest to medical researchers and clinicians, viz., Biochemical Interpretation of Antibiotic Resistance of Micro-organisms (by di Marco); Cross-resistance among Four Antibiotics: Neomycin, Paromomycin, Kanamycin and Streptomycin (by Finland); Combined Antibiotic Action (by Jawetz); Modes of Action of Antibiotics (by Hahn); and Some Biochemical Foundations in the Search for Anticancer Antibiotics (by Ganse).

The papers give a fair idea of the trends of research on the biochemistry of antibiotics. The search for antibiotics in different laboratories of the universities and industry has resulted in the isolation of antibiotics of various structures which may be grouped as those derived from the main metabolic pathway of proteins, fats, carbohydrates and those from purine metabolism. Of late the interest of antibiotic research has shifted from the purely structural to the more biochemical aspects. The biosynthetical studies of antibiotics have led to the understanding of the mechanism of biosynthesis of penicillin; tracer experiments, isolation and knowledge of enzyme systems may reveal the mechanism of biosynthesis of other antibiotics.

The mode of action of an antibiotic, of which definite progress has been made in the penicillin field only, shows that in the case of penicillin, its antibacterial action is due to its interference with the synthesis of cell wall material.

When a mixture of two antibiotics is acting on bacteria the effect can be greater than that of any one of the single antibiotic. It is found that no one pair of antibiotics is consistently synergistic under all circumstances. It is essential to establish for each individual case the best antibiotic combination. Although the synergistic action is seen among penicillin, streptomy-

cin, bacitracin, neomycin and polymixin, yet it is not observed among chloramophenicol, erythromycin, novobiocin and tetracyclines. Of course the antibiotic combination therapy may retard the emergence of resistant strains, and that has been demonstrated in the treatment of tuberculosis.

It is not understood why antibiotics, since they act against, bacterial infections, would act against cancer, which probably resembles bacterial infections only in growth. In cancer, it is necessary to arrest the growth of tissue cells which have become abnormal and have no control over their growth and destroy neighbouring normal cells. Such cancerous cells unfortunately show no significant difference from the normal cells. The antibiotic if it is to act against cancerous cell must of course differentiate between these and normal cells from which the cancerous cells originate. Antibiotics having cytostatic action do not necessarily act on cancerous cells. It seems, therefore, that the chances of finding an anti-tumour antibiotic are very remote.

The book deals with the various topics of interest to present-day medical research and is invaluable to all concerned with research on the biochemistry of antibiotics.

A Pocket Book of Proprietary Drugs—By Alistair G. Cruikshank, F.R.C.P.E., and Cornelius Stewart, L.R.C.P., & S.E., M.P.S. 1959, E. & S. Livingstone Ltd., Edinburgh. 6" ×4½", pp. 236, price 10s. 6d.

This small book contains useful information about important proprietary drugs. The volume is well produced and can be profitably kept in the doctor's bag. The proprietary drugs are only of British firms or of firms having branches in the United Kingdom. As such no products of Indian firms have a place in the volume. At the end of the book, there is a list of additional recent products and it will be a job for the compilers to keep pace with the race for manufacture of really good drugs. For the dosage of certain products, the compilers have suggested the consultation of relevant literature. In the next edition of the book, this should be avoided. In the index for different products, the page in which a particular product has been described, should have been given for the convenience of the readers.

A Handbook for Dissectors—Edited by J. C. Boileau Grant, Professor Emeritus of Anatomy and Curator of the Anatomy Museum, The University of Toronto. (Formerly ed. by J. C. B. Grant and H. A. Cates). 5th edition, 1959; Ballière, Tindall and Cox, Ltd., London. 4¾" ×7¾", pp. 439, 135 figures, price $5.00.

The usefulness and popularity of this book are indicated by the fact that since its first publication in 1940, it has gone through several reprintings and new editions. In the present edition the instructions as to the procedures for dissections so that the dissector can help himself has been dealt with in greater details. Many new diagrams have been added. The new international nomenclature has been employed in place of B.N.A. and where differences between the two nomenclatures are considerable, the old synonyms have been indicated in brackets. Throughout the book have been stressed big things and fundamentals that are useful rather than details. The text is lucid, illustrations superb and binding excellent. The book is strongly recommended for undergraduate medical students for whom it is intended.

The Arterial Wall (Aging, Structure and Chemistry)—Edited by Albert I. Lansing, A.B., PH.D., Chairman, Department of Anatomy, University of Pittsburgh, School of Medicine, Pittsburgh, Pennsylvania. (Sponsored by the Gerontological Society, Inc.). 1959, The Williams & Wilkins Company, Baltimore. 6" ×9", pp. 259, 16 plates, price $7·50.

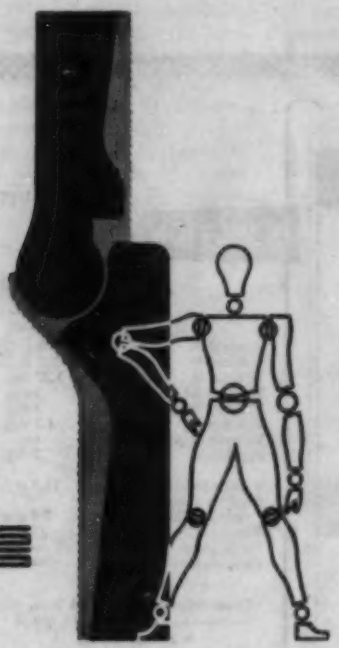
This outstanding monograph is the outcome of intense curiosity of a layman the late Mr. Forest D. Dorn who had the farsight to appreciate the far-reaching importance of such a study of an organ system which pervades the entire body and determines its welfare from cradle to grave. For this he supported the project by a grant to the Gerontological Society for the study of this vital anatomical unit on its dynamic aspect.

This monograph is divided into nine sections each dealing with one separate component of the structure, biochemistry and metabolism with reference to vascular ageing and atherosclerosis and the relation between them. The book ends with an excellent summing up emphasising the major points of interest. Each group of sectional contributors has compiled material from all available up-to-date research papers with their references at the end of the chapter. The contributors have tried to present how much is known at present and what remains to be known. They have also raised thought-provoking questions that are certain to inspire and excite an enquiring mind to accept the challenge for elucidation of the problem in this none-too-easy field. The book will certainly be a precious addition to the book-shelf of every medical teacher irrespective of his primary field of interest and is certainly a treasure to workers already probing the different aspects of this important region of study.

Nierenkrankheiten : Physiologie, Pathophysiologie, Untersuchungsmethoden, Klinik und Therapie—Von Prof. Dr. H. Sarre, Freiburg/Br. 2., verbesserte Auflage, 1959. XXIV, 555 Seiten, 134 Abbildungen, G.-8° (Georg Thieme Verlag, Stuttgart), Ganzleinen DM 59.

Renal Diseases : Physiology, Pathophysiology, Examination Methods, Clinic and Therapy—By Prof. Dr. H. Sarre, Freiburg/Br. 2nd improved ed., (in German). DM 59.

The first edition of this excellent book was published a little over a year ago (1958) and reviewed in this column on May 16, 1958. Although the text has been increased only by 15 pages, 16 new illustrations and more than 100 references have been added. Almost every chapter has been revised and brought up to the



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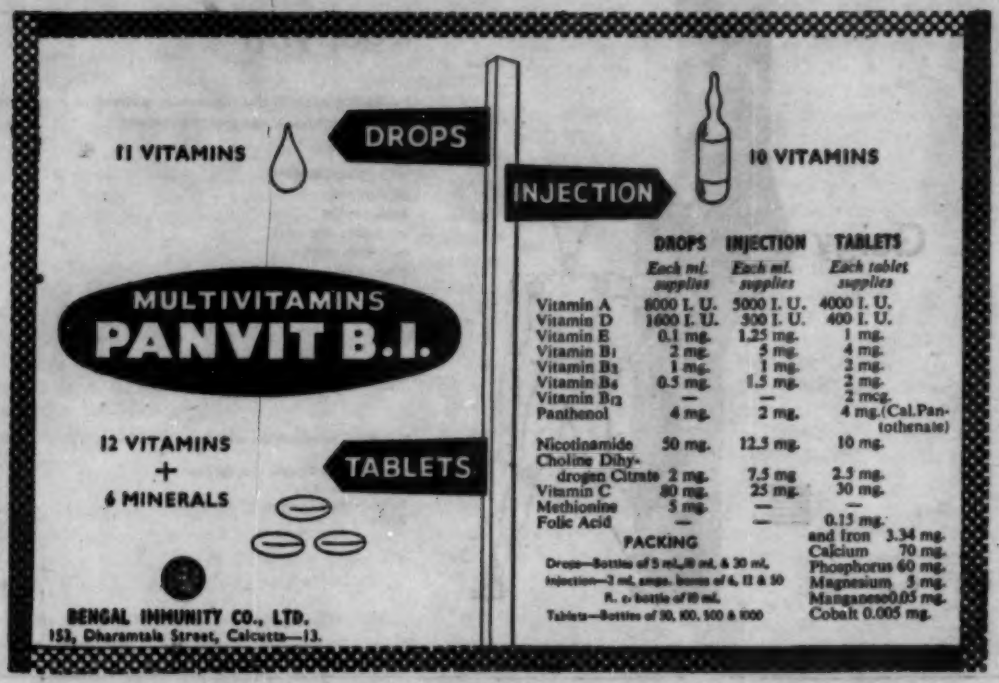

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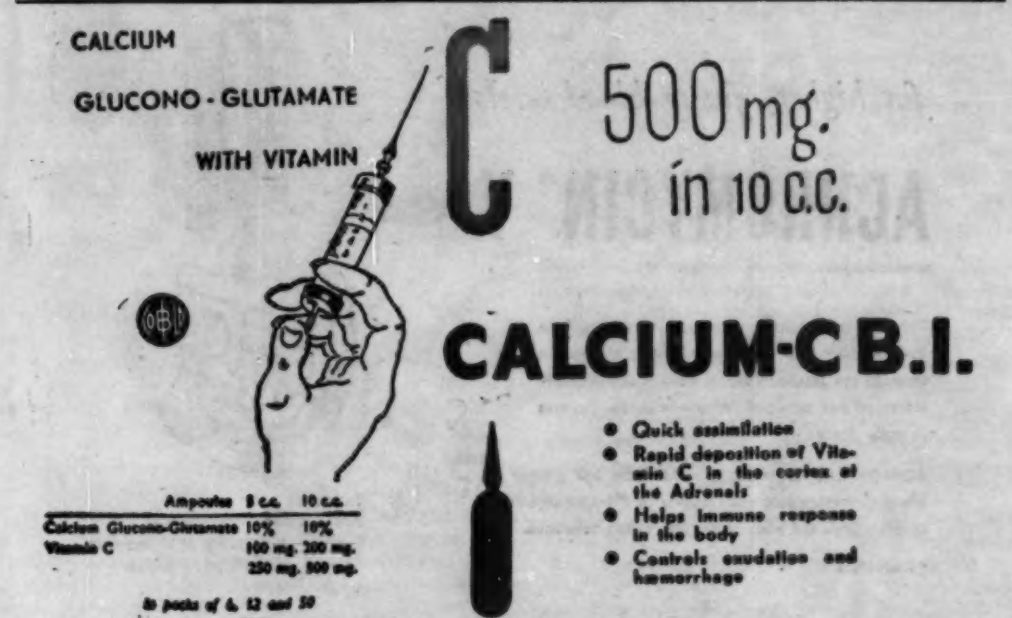
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very latest position of scientific and clinical development. The intrarenal processes of urinary concentration, hormonal regulation, pathogenesis of nephrosis, substitution of improved laboratory methods, classification of renal diseases are some of the revised items; syndromes that only recently came to the forefront have been added, such as idiopathic hypercalciuria, oxalosis, glycin- and xanthinuria. Dietetic, medicinal (hydrochlorothiazid has already been included) and treatment by dialysis have been revised and new tables of normal values, diet and differential diagnosis further enhance the usefulness of this work, for which it is hardly possible to find an equal in English literature.

Notes on Injections for Nurses—By T. H. White, M.B., CH.B., D.T.M. & H. 1959, John Wright & Sons Ltd., The Stone Bridge Press, Bath Road, Bristol 4. 7¼" x 4¼", pp. 32, price 2s. 6d., postage 2d.

The small book contains useful information on the subject. Though the technique of giving injections is learnt in the hospital wards by watching how the injections are being given by another person and later modifying the technique according to one's own experience, this small treatise will be helpful in learning the different practical aspects of giving injections.

It should be impressed that the most common and important cause of pain while the injection is being given is a blunt or thick needle and that rectified spirit is not the proper material to sterilise syringes or the skin of patients.

Lehrbuch der Krankengymnastik (In 4 Bänden)—Herausgegeben von K. Lindemann, Heidelberg, H. Teirich-Leube, Freiburg/Br., W. Heipertz, Bad Kreuznach. Band I: Einführung in die Krankengymnastik. Physiologie. Krankheitslehre. Grundlagen und Grundtechnik der Krankengymnastik und Massage. Bearbeitet von E. v. Dietze, Bad Oeynhausen, W. Hardegg, Heidelberg, J. Harff, Hamburg, W. Heipertz, Bad Kreuznach, K. Lindemann, Heidelberg, B. Lück Heidelberg, H. Teirich-Leube, Freiburg/Br. 1959. XII, 328 Seiten, 222 Abbildungen, Gr.-8°, (Georg Thieme Verlag, Stuttgart), Ganzleinen DM 32.— (Staffelpreis ab 10 Expl. DM 27.20). Jeder Band ist einzeln käuflich.

Text Book of Medical Gymnastic in 4 volumes—Ed. by K. Lindemann, Heidelberg; H. Teirich-Leube, Freiburg/Br., W. Heipertz, Bad Kreuznach. Vol. I. Introduction to medical gymnastics, physiology, pathology, principles and basic technique of medical gymnastics and massage by 7 experts (names see above). 222 illustrations. Each volume available separately (in German). DM 32.

Medical gymnastics and physiotherapy have gained a secure position in almost all sections of medical practice. A complete survey of all the facts required for teaching and training physiotherapists proved, therefore, necessary. The present encylopaedic work is intended to fill this gap.

The first volume starts with an outline of physiology (100 pp.), followed by 40 pages devoted to general pathology and the diseases of the locomotor system. A few pages on first aid and dressing, and on history of medicinal gymnastics precede the chapters dealing with the subject-matter proper. They give detailed information on general medical gymnastics (84 illustr.) and its basic techniques (55 illustr.), such as positioning, movement of single joints, active movement therapy and special methods, therapeutic pool treatment and, finally, 60 pages with 59 illustrations on massage. This volume takes the physiotherapist from the scientific foundations of his craft to some of the most important technical methods. They are so well described and so fully illustrated that teaching and being taught with such a book to help should be a pleasure. One can only hope that the remaining 3 volumes will keep to the same high standard regarding contents and production.

Psychiatrische und Nervenklinik: Krankenvorstellungen—Von Prof. Dr. K. Kolle, München. 1959. XVI, 252 Seiten, Gr.-8°, (Georg Thieme Verlag, Stuttgart), kartoniert DM 19·80.

Psychiatric and Neurological Clinic: Case Demonstrations—By Prof. Dr. K. Kolle, Munich (in German), DM 19·80.

The author publishes here 17 lectures which he delivered as professor of psychiatry and neurology, and director of the neurological clinic, University of Munich. They consist of 187 case demonstrations, i.e., the verbatim record of the conversations between the professor, whose wife acted as stenographer, and the patients, followed always by the professor's commentary and diagnosis. As each lecture lasts in fact 2½ hours and every time are shown: first a neurological case, then one suffering from a cerebral disease with psychic as well as neurologic signs and symptoms and, finally, psychoses and neuroses, the material is rich and varied. Although the patients are not being shown systematically but according to the most suitable material available, the lectures have been arranged under the following headlines: invasion of the personality by disease; the person is deprived of self-control; damages, invading the stages of development, prevent maturation of the personality; the world has been transformed; the ego has been alienated; the ego entangled in itself; the organs required for carrying out personal acts refuse to work; strike of the depth-personality; mind-soul-body as a medical problem; the effector organs stop working; man gives in; fate has to be overcome; man as a member of society; fits; suicide; the physician as a helper; the physician as a researcher.

In Germany or Austria where for many years neuropsychiatry has been taught in considerable detail throughout the clinical years and every student has to pass a difficult examination in these subjects before graduating, this work will become a most welcome textbook. Here, where psychiatric as well as neurologic training is rudimentary only and experts are few, a translation of this book that imparts all essentials in a simple and didactically perfect way, free from modern psychologic slang, should be of the greatest value to practitioners and experts alike.

OBITUARY

Dr. K. Damodaran

Dr. K. Damodaran, Superintendent, Central Hospital, Asansol, was born on 12-10-1907. After his early education at Varkala (Kerala) he obtained his B.A. degree in 1928. He passed the M.B.B.S. examination of the Madras University in 1934. He was granted free studentship by the Government of Madras for the whole course of M.B.B.S. for his scholarly brilliance.

His first appointment was as Medical Officer in Maldives after which he worked as House Physician in Government General Hospital, Madras, from 1936. He left for England in 1938 for further studies in Medicine and took his L.R.C.P. (Lond.) and M.R.C.S. (Eng.) in 1939 and M.R.C.P. (Edin.) in 1940. He practised in London and in February 1941 became a Commissioned Officer of the R.A.M.C.

The 45 B.G.H. to which he was attached as a Lieutenant moved from London to the isle of Malta. He was in Malta for 2 years experiencing the continuous bombing by the Germans. It was there that he did considerable research in infective hepatitis on which he wrote many articles in the *British Medical Journal*.

The War Services took him to Egypt and the Middle East also where he was promoted to the rank of Captain. In 1944 he was transferred to India and posted at Bombay. He was then posted in succession at Madras, Deolali, Jalahalli and Avadi. By 1945, he rose to the rank of Major in the R.A.M.C. He was recalled to U.K. and placed in charge of a Unit in Yorkshire with the Military Hospital at Catterick Camp.

Major Damodaran was research-minded and the War had given him much scope for research. He received Defence Medal, Africa Star and other Service Medals in appreciation of his commendable services in the Army.

The British Government appreciated his services so much that he was offered a Permanent Commission in the R.A.M.C. His patriotism and long-cherished desire to serve the masses of his motherland compelled him to decline the offer of permanent commission in the R.A.M.C. and instead he urged for his release. He was released from the British Army in 1950. On release he was on the staff of the Royal Infirmary under the Edinburgh University for 6 months and took his diploma in Tropical Medicine and Hygiene of that university in 1951. Thereafter, he took up another appointment as M.O. In-Charge of the Medical Section, Ministry of Pensions Hospital, Musselburgh, Scotland, for a short while, and then returned to India.

He was appointed Specialist Physician for Central Hospital, Dhanbad, in 1952. In 1954 he was also appointed as the Superintendent of the Central Hospital, Asansol. As Superintendent of this Hospital, he worked untiringly for the improvement of the institution.

DR. K. DAMODARAN

Also he had under his charge the administrative control of two Regional Hospitals and one TB Clinic in Raniganj coalfield and two Regional Hospitals in Madhya Pradesh coalfields. He also had the supervision over the treatment of TB patients at the domiciliary treatment centres.

Dr. Damodaran was very popular wherever he worked and lived and was known for his gentle behaviour towards his patients.

He was the President of the Asansol Branch, I.M.A., for two consecutive terms 1958-60.

Dr. Damodaran passed away on 12-1-60.

XII ORISSA STATE MEDICAL CONFERENCE, 1960, BHUBANESWAR

The 12th Orissa State Medical Conference will be held at Bhubaneswar on the 19th and 20th March 1960. Dr. Subal Chandra Das has been elected as the Chairman, Reception Committee and Dr. G. D. Mahapatra and Dr. K. C. Roy as Organising Secretaries of the Conference. Further information may be had from Dr. K. C. Roy, New Capital, Bhubaneswar, Orissa.

X ASSAM STATE MEDICAL CONFERENCE, 1960, JORHAT

The 10th Assam State Medical Conference could not be held on 16-18 January 1960 as announced previously. It has been finally decided to hold the conference on 20-22 February 1960 at Jorhat.

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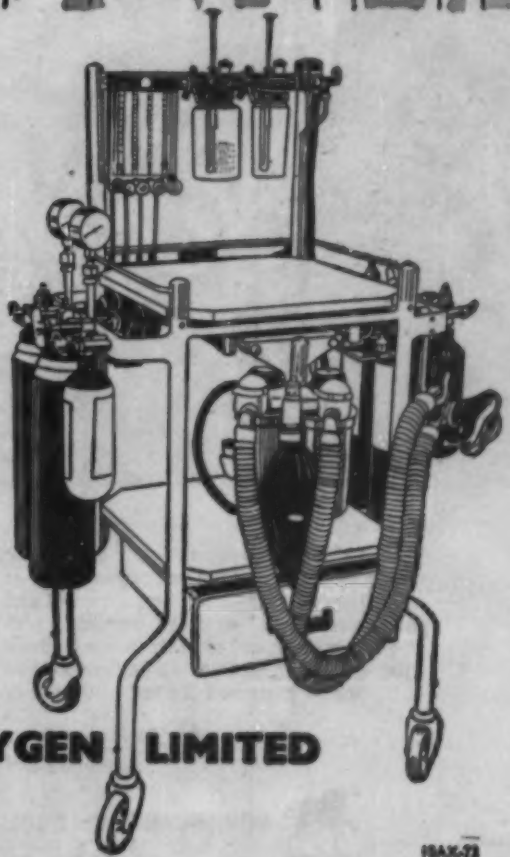

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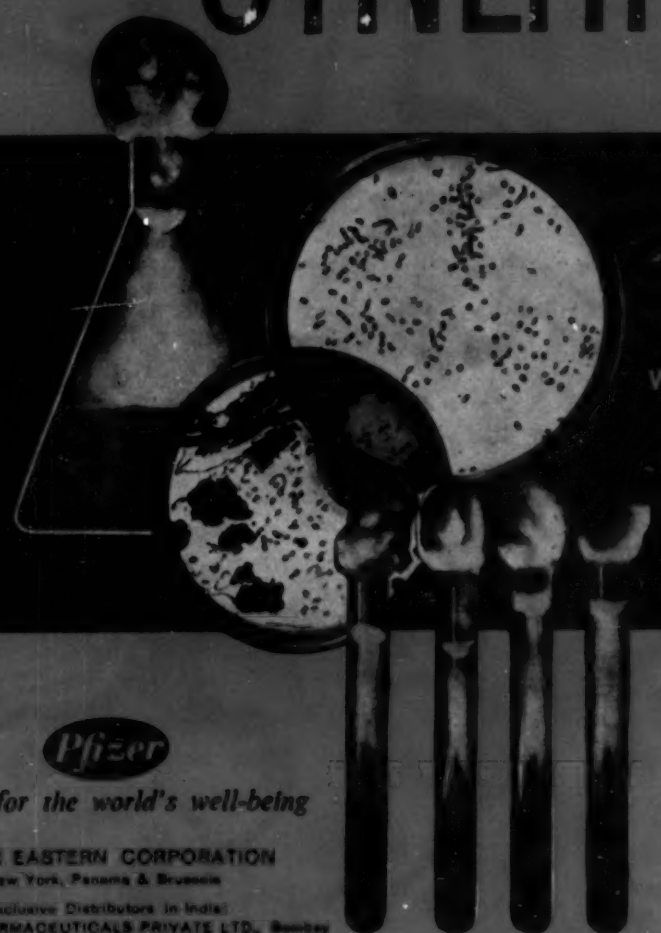

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1. Russek, H.I.: *Am. J. Cardiol. 3:547 (April) 1959.*

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